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An Address.[1]

THE RHEUMATIC CHILD.

By E. BRITTEN JONES, M.B. (Adelaide), M.R.C.P. (London),
Retiring President of the South Australian Branch of the British Medical Association.

One of the most difficult problems confronting the holder of the office I am relinquishing this evening is the choice of a subject for his presidential address. When I recall the many admirable addresses that have been delivered on this occasion during recent years and realize that most of these dealt with subjects of ethical, medico-political and historical interest, I feel that some few words of explanation are demanded of me when I say that I have chosen a scientific subject for my address tonight. I propose to discuss briefly the rheumatic child.

[1] Read at the annual meeting of the South Australian Branch of the British Medical Association on June 27, 1934.

During recent years in Great Britain increased and increasing thought and experiment have been devoted to the problem of the rheumatic child, and it is a source of gratification to us as members of the British Medical Association that the impetus given to rheumatic research and treatment is in no small measure due to the science committee of the parent Association in England, and if it is my good fortune to arouse interest in the problem as it confronts us here in South Australia, the aims and objects of the Association may in some small way be furthered.

HISTORY.

Rheumatic fever was first described as an entity and given the name of rheumatism by Guillaume Baillou in a publication of 1762, the name itself suggesting association with inflammatory affection of the upper respiratory passages.

The next name of importance, especially as regards England, is that of Thomas Sydenham, whose description of the symptoms shows the same

careful observations that characterize his classical treatises on gout and chorea.

It generally originates in some such cause as the following: The patient has been heated by either some over-violent exercise or by some other means and has taken cold upon it: The sad list of symptoms begins with chills and shivers; these are followed immediately by heat, disquietude, thirst and the other concomitants of fever.

One or two days after this (sometimes sooner) the patient is attacked by severe pains in the joints, sometimes in one and sometimes in another, sometimes in his wrist, sometimes in his shoulder, sometimes in the knee—in the last joint oftenest. This pain changes its place from time to time, takes the joints in turns and affects the one that it attacks last with redness and swelling.

The next worker in this field whose writings have come down to us is Richard Morton (1637-1698). After taking orders in the Church, he turned towards the study of medicine at the age of twenty-five and took his doctorate in medicine at Oxford at the age of thirty-three; he subsequently became a Fellow of the Royal College of Physicians and physician to the King. Although his most important work was in connexion with consumption and wasting diseases generally, and published in his "Phthisiologia", he also contributed to the literature on gout and rheumatism, especially from the therapeutic standpoint. He described his treatment in one case as follows:

I endeavoured to satisfy the present indications with Bleeding, a Blister, an Anodyne Draught, Tincture of Roses, a Pectorial Linctus and a Pearl Julip; and indeed all the symptoms going off at the end of the fit, when I came to see my patient the next day, I reckoned the Distemper was overcome and removed with those things I had ordered, till at length by the return of the Fever and Rheumatism the next night, I plainly understood how vainly I had hitherto flattered myself.

Such therapeutic candour is indeed refreshing.

John Haygarth, one of the most prominent medical figures of his time, and a Yorkshireman by birth, published his "Clinical History of the Acute Rheumatism" in 1805. He stated that:

Exposure to cold and moisture is the principal cause of acute Rheumatism; for this reason we cannot be too minute and diligent in our endeavours to investigate the circumstances in which this enemy of mankind produces such injurious effects.

Another outstanding name in connexion with our subject tonight is that of William Charles Wells, who was born in South Carolina in 1757, the son of Robert Wells, who had immigrated from Scotland. His father was an ardent loyalist and made his son wear a cotton coat and a blue bonnet so that he might not be mistaken for an American. Wells junior decided to remove all possibilities of such an unfortunate happening by proceeding to England, doing his medical course in Edinburgh and ultimately becoming physician to Saint Thomas's Hospital. In 1810 he published what is perhaps the earliest account of the cardiac complications of rheumatic fever; in one of his cases he noted that:

Many of the superficial muscles in this patient were studded with numerous small hard tumours, an appearance I have observed only in one other person, a thin and feeble man of forty-one years old, who also laboured under rheumatism.

I think this must be one of the earliest references to the rheumatic nodule.

But the clear-cut association between endocarditis and rheumatic fever was placed on a sure foundation by Jean Bouillaud, of Paris, in 1836. He became professor of medicine at Paris at the early age of thirty-six, and so popular was the appointment that his students carried him on their shoulders in triumph around his hospital. To quote a passage from his works:

I soon discovered that an acute affection of the heart in cases of acute articular rheumatism associated with violent fever, was not a simple accident, a rare, or as it were fortuitous complication, but in truth the most usual accompaniment of the disease.

I do not propose to trace the development of knowledge concerning our subject down to modern times, but I think references should be made to the classical paper which Sir Thomas Barlow contributed to the fifty-first annual meeting of the Association, held at Liverpool, in 1883. This paper, entitled "Notes on Rheumatism and its Allies in Childhood", is a masterly exposition of the subject, and the intervening fifty-one years have added little to our knowledge of the subject. I can recommend its perusal to those of you especially interested in rheumatism of childhood. Barlow drew attention to the association with tonsillitis:

First let us note the frequent occurrence of tonsillitis, either ushering in the fever or preceding it by a week or two.

After discussing the nature of the rheumatic nodule, he goes on to say.

If it be permitted to speculate as to the homology of these nodules I should like to suggest that, considering their structure and natural history, they may perhaps correspond with the inflammatory exudation which forms the base of a cardiac vegetation, on the top of which latter a cap of fibrine may or may not become deposited.

Enough has been said to show the wealth of knowledge that has been handed down to us, and we as a profession would prove unworthy of our inheritance if we failed to pursue still further the investigation of rheumatic fever by modern methods, especially here in South Australia, under conditions which I hold to be eminently favourable to the elucidation of certain problems connected with the disease.

At the annual meeting of the Association in Portsmouth in 1923, a discussion, which was opened by Dr. Reginald Miller, took place on the ætiology and treatment of heart disease in early life. At the end of this discussion it was resolved:

That the Council of the Association be recommended to appoint a special committee to consider the best steps to be taken to combat the grave menace to the community arising from cardiac diseases.

As a result a subcommittee was appointed and the terms of reference were as follows:

To enquire into the steps which have been and are being taken in relation to the prevention, detection and treatment of cardiac disease in children, and to make proposals in connection therewith.

The members of the subcommittee were: Sir Humphrey Rolleston (chairman), Dr. G. A. Allan, Dr. Carey F. Coombs, Dr. F. J. Poynton, Dr. A. P. Thompson, and Dr. Reginald Miller. Sir Thomas

Lewis was subsequently coopted. The report of the subcommittee, issued some two and a half years later, stressed the need for thorough organization of effort for the prevention and after-care of this disease; without entering into any detail it may be said that it represented a very valuable addition to our knowledge of the disease and indicated the direction that further research should take.

Investigation of the Disease in South Australia.

Incidence.

With regard to the incidence of rheumatic disease in South Australia at the present time, no definite figures are available, and I must perforce fall back on the records of the Adelaide Children's Hospital. At the outset of this inquiry I went through the case notes of all children admitted to that institution from 1928 to 1933 inclusive—a period of six years. In this inquiry only cases of rheumatic fever, chorea and rheumatic carditis were included, and other possible rheumatic manifestations, such as tonsillitis, *erythema nodosum* and phlyctenular conjunctivitis were not included, although I think that the large majority of both these latter two clinical entities rest on a tuberculous basis. The numbers of admissions were as follows:

1928	30 cases
1929	37 cases
1930	42 cases
1931	38 cases
1932	37 cases
1933	44 cases
Total	218 cases

This makes a total of 218 cases. The following statement shows the average stay in hospital and the total cost and the average annual cost to the Children's Hospital.

Total number of cases	218	
Total number of days in hospital	19,962	(2,851 weeks)
Average number of days per patient	91	
Cost for 2,851 weeks at £2 3s. 6d. per week	£6,200	
Average yearly expenses (for years 1928 to 1933 inclusive)	£1,033	

Places of Origin of Rheumatic Fever.

The next point to be investigated was the place of origin of these rheumatic cases. It was found that of the 218 cases investigated, 194 came from the city and suburbs and 24 from the country. Further investigations of the country cases disclosed the interesting fact that 50% of these came from the hills districts surrounding the city of Adelaide. This interesting distribution is shown below.

Hills Districts.	Other Country Districts.
Barossa	Berri
Bradbury	Murray Bridge
Blackwood	Monteath
Campbelltown	Winkie
Cherryville	Salisbury
Echunga	Dry Creek
Halbury	Gawler
Meadows	Gilles Plains
Mount Compass	Port Lincoln
Mount Pleasant	Ceduna
Scott's Creek	Gladstone
Yankalilla	Oodnadatta

These figures certainly suggest that rheumatic disease is common in the hills districts and not uncommon along the low-lying districts in the Murray Valley, but that it is uncommon in the northern parts of this State. The one case from Oodnadatta is perhaps unfortunate in this connexion; but I do not propose to allow it to divert me from making the broad statement that I think further investigation would show that rheumatic diseases in children are very uncommon in the more northern parts of this State. The work of Coburn in America suggests this probability; he showed that rheumatic children sent from New York to the warmer climate of Porto Rico remained free from any rheumatic manifestation while they remained in the West Indies, but on their return to New York recurrence was the rule.

Of the cases occurring in the city and suburbs, 194 in all, 23 came from the city; the remaining 171 cases were widely distributed, and no special facts of import arise from their study other than that rheumatic disease appears to be especially prevalent in the Port Adelaide district and in Norwood, whereas in the northern and southern suburbs there is a considerably lower incidence as far as the admissions to the Children's Hospital disclose.

The Serious Cases.

The next line of investigation was to ascertain if the study of the more serious cases disclosed any suggestive facts.

The criteria of serious cases were arbitrarily put down as: death, pericarditis, presence of nodules, gross cardiac enlargement, aortic disease. Using this as a basis it was found that there were 33 cases that could reasonably be regarded as grave.

Death occurred in 22 of these 33 cases. While various factors contributed to this unfortunate termination, it is noteworthy that the immediate cause of death was infective endocarditis, either acute or subacute, in nine cases.

Of these 33 grave cases 23 were from the city and suburbs and 10 from the country.

It would be injudicious juggling with figures to reduce these findings to percentages of case admissions from city and suburbs and country respectively, as it is reasonable to suppose that many more mild cases would find their way to the Children's Hospital from city and suburban areas than from country districts; but I personally have been most impressed with the fact that the country cases are definitely of a more severe and fulminating type.

Nodules are generally supposed to be most unusual rheumatic manifestations in Australia; but I would question this idea. In severe carditis they are not an uncommon finding and were present in nine cases in the present series, five of which came from the country; the child with a multitude of nodules is practically always a country child.

Why is the country child prone to these manifestations of severe rheumatism?

We know the ravages the tubercle bacillus is likely to play on virgin soil; perhaps the country child's defences against streptococcal infection have not reached the efficiency we meet with in the urban child.

As I have spoken of streptococcal infection, it is necessary to add that while the ætiology of rheumatism in childhood is not yet proved, evidence is gradually accumulating pointing to a streptococcus playing a large part in causation; a discussion as to whether the heart, brain and joint manifestations are a direct result of infection of these tissues with streptococci or whether they are rather of the nature of local allergic manifestations consequent on the throat infection is rather beyond the scope of this paper.

Cardiac Damage.

The next investigation undertaken was an attempt to discover how many of the 196 children who left hospital did so with a damaged heart as a result of the rheumatic infection.

I found it difficult to establish criteria of cardiac damage from a series of case notes; it was impossible to assess the damage to the cardiac muscle, and I had to rely on the clinical evidence of involvement of the valves. I take it that the presence of a diastolic or a presystolic bruit in the mitral area, in addition to the systolic bruit, can be taken as definite evidence of valve damage. When one comes to decide on the significance of a murmur, purely systolic in time, one is on rather difficult ground. While the loud, rasping, systolic murmur frequently heard can leave one in very little doubt as to its organic origin, the faint systolic bruit heard at the time of the patient's discharge from hospital may or may not mean valve damage. However, an attempt has been made to obtain some idea of the valvular damage in these cases under review. The figures are set out below.

Patients who died	22
Total number of patients who left hospital ..	196
Patients with no evidence of valvular disease	97
Patients with definite evidence of mitral stenosis (a diastolic or presystolic bruit in the mitral area in addition to a systolic bruit)	37
Patients who left hospital with a mitral systolic bruit of varying intensity	62

So, in addition to the 22 fatal cases, I think it is a conservative estimate to state that some 70 patients were discharged with damaged hearts and a large number of these were condemned to a life of invalidism or at least a considerable reduction in their earning capacity.

Mitral Stenosis in Women.

A further investigation was carried out with a view to learning if the case records at the Children's Hospital gave any evidence presaging the undoubted preponderance of mitral stenosis met with in adult females. It has been my impression for some time that we meet with the early signs of stenosis more commonly in the female child.

Table I shows the results of this inquiry.

TABLE I.

Showing the sex incidence of mitral stenosis and mitral systolic bruit among children leaving hospital after recovery from acute rheumatism.

	Definite Mitral Stenosis.	Mitral Systolic Bruit.	Total.
Boys	11	37	48
Girls	25	26	51
Total ..	36	63	99

Features of Clinical Interest.

The studies of the case histories used in the preparation of the figures I have placed before you tonight have disclosed some matters of clinical interest which I should like briefly to touch upon.

Seven of these rheumatic children were sent into hospital complaining of abdominal pain, apparently of considerable severity, in some cases associated with vomiting. In the majority of these the results of clinical examination stayed the surgeon's hand; but two children were subjected to appendicectomy, the diagnosis of rheumatic fever being subsequently established by the development of joint swellings which responded to salicylates. The diagnosis of rheumatism must therefore be considered in any case of indefinite abdominal pain in children, and careful examination of the heart and joints may save an unnecessary laparotomy.

During the past few years increased attention has been focused on the association of tonsillitis and rheumatism in childhood, especially on the apparently latent interval between the throat symptoms and the onset of joint or heart manifestations. You will remember that Sir Thomas Barlow drew attention to this particular point. In this connexion Glover and Griffith, Collis, Shelton, and Schlesinger have brought forward a considerable volume of evidence that the rheumatic symptoms, either initially or representing a relapse, follow the tonsillitis at an interval varying from ten to twenty-one days; in fact, very much the same time relationship exists as has been found to obtain in acute nephritis in children following streptococcal infection of the throat. Without entering into details it can be broadly stated that the study of the histories of the cases under review this evening amply confirms these findings. In 1923 the subcommittee previously mentioned reported its findings on the value of tonsillectomy in the rheumatic child; these conclusions were necessarily indefinite in view of the fact that they were based on the results of a comparatively small number of cases—133. However, it was suggested that the tonsillectomy could be performed with safety at a comparatively short interval after the subsidence of the acute symptoms, and that tonsillectomy in rheumatic children, while lessening the arthritic and cardiac recurrences, apparently increased relapses, in which chorea was the manifestation of the rheumatic infection. My opinion is that this finding should be received with a considerable degree of caution, and much further investigation is

needed before we can rightly assess the value of tonsillectomy in such cases.

I might mention that in three cases under review a relapse or an initial manifestation appeared to be definitely related to the operative interference.

D.C., aged eight years, a child known to be rheumatic, developed a relapse of carditis three weeks after tonsillectomy. Two other patients developed apparently primary attacks of rheumatic fever five days after removal of tonsils, one patient being affected with subsequent valvular damage and the other developing myocarditis.

I would say that tonsillectomy should only be considered—and I use the word "considered" advisedly—when the rheumatic infection is apparently quiescent, as judged by pulse rate, red blood cell sedimentation rate, and the other means we have of assessing the activity of the rheumatic process. In this opinion I know I am not in accord with the current medical opinion.

Facilities for Treatment.

The facilities that exist at present in Adelaide for the study and institutional treatment of the rheumatic child naturally come more or less entirely within the ambit of the activities of the Children's Hospital. Until fifteen months ago rheumatic children were treated in the wards. As a result one was confronted with a disappointingly high rate of relapse, especially of carditis. This was, I feel sure, due to repeated fresh streptococcal throat infections inseparable from a children's ward, with its visitors and its frequent new admissions. Some patients were then sent to Mount Lofty to convalesce with equally unsatisfactory results. However, some fifteen months ago the James Brown Memorial Trust very kindly came to our assistance and placed some beds at Escourt House at our disposal for the reception of rheumatic cases; by judicial management the number of beds has gradually been increased to about fifteen. This number I hold to be inadequate, and I would say that an institution with a capacity of at least thirty beds is necessary to deal adequately with the problem.

I have been most impressed with the results obtained at Escourt House, the infrequent incidence of relapse being especially noteworthy. The Education Department has cooperated in providing educational facilities for these children, whose future means of livelihood is likely to be considerably restricted.

The Children's Hospital has further established a rheumatic clinic in connexion with its outpatients' department, and this, I think, insures a more satisfactory follow-up of the child's progress when he leaves hospital or Escourt House.

There is, however, need for closer cooperation between the Children's Hospital and the Adelaide Hospital to insure suitable supervision of the rheumatic child passing into adolescence. Some adequate arrangement could no doubt be arrived at. I would only stress the desirability of all such patients being passed on to one clinic, preferably to a special clinic at the senior institution. By supervision in these adolescent years the youth or girl bearing the scars of childhood rheumatism may be safeguarded from undertaking an occupation calculated to throw undue strain on a damaged heart.

Recommendations.

The figures I have shown you tonight prove, I think, that rheumatic infection is prevalent in South Australia; and while I have confined my remarks to the immediate results of such infection, the remote effects thereof would show even more convincing proof of the need for some concerted effort to combat the disease as we meet it in South Australia. How that effort should be inaugurated is a matter for consideration and debate. But I can imagine no more fitting gesture from this Branch to the parent Association than that your Council should appoint a subcommittee to investigate childhood rheumatism as it exists in South Australia—a subcommittee that should furnish a report on the lines of the report furnished by the subcommittee of the home Association.

This may seem a somewhat ambitious project; but from the figures put before you tonight we have ample material for investigation; furthermore, our varying climatic conditions, the undoubted frequency of rheumatic infection in certain areas, the apparent severity of cases coming from sparsely populated country districts, the feasibility of making it a notifiable disease, and the centralization of a considerable proportion of the acute cases in one institution should provide a fertile field for investigation; and the findings of such a subcommittee could not fail to add to the sum total of our knowledge of rheumatic disease in children.

ADENOHYPOPHYSEAL DYSTROPHIES OF INFANCY AND CHILDHOOD.[1]

By LORIMER DODS, M.B., Ch.M. (Sydney),
Honorary Relieving Assistant Physician, Royal Alexandra Hospital for Children, Sydney.

I PARTICULARLY appreciate the honour of being asked to address you on this most fascinating borderline of general medicine and pædiatrics. I am acutely conscious of my relative ignorance of this difficult subject and realize that my brief paper, with its obvious shortcomings, must prove a rather lame and uninteresting prologue to the concise and dramatic exposition of similar problems which you are about to hear from Professor Lambie.

I have attempted to limit my remarks to disturbances of adenohypophyseal function and have

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 28, 1934.

avoided as far as possible any discussion of the problems associated with the confusing neurohypophyseal mechanism. Before discussing some of the clinical syndromes associated with such disturbances, I propose to attempt an extremely brief outline of our present knowledge of certain aspects of the physiology of this lobe of the pituitary gland. It is presumed that the adenohypophysis secretes: (i) a growth factor which controls skeletal and general body growth; (ii) sex or gonadotropic factors which, besides their other functions, control the development of the genitals and of the secondary sexual characteristics; (iii) a thyreotropic factor which controls to some degree the function of the thyreoid gland; (iv) an adrenalotropic factor which exerts some control over the functioning of the adrenal cortex; (v) a lactogenic factor which plays some part in controlling the development and secretion of the mammary glands; (vi) a factor which possibly influences carbohydrate metabolism.

The adenohypophysis is composed of chromophile and chromophobe cells, the chromophile cells being designated as acidophilic or basophilic, according to their affinity for acid or basic dyes. Animal experimentation and clinical studies have proved that the acidophile cells elaborate the growth factor and that the basophile cells probably produce the sex or gonadotropic factors. The actual function of the chromophobe cells remains unknown; overgrowth of these cells may produce disturbances of pituitary function by mechanical pressure.

In the following discussion of clinical syndromes I have adopted and have endeavoured to follow the above simple and rather primitive conception of anterior pituitary physiology which has been embodied in the accompanying table. This table is in most respects a modification of a classification suggested by Lambie and Wiesener;[1] it includes the several metabolic factors under one heading and affords a convenient means of reclassifying on an approximate physiological basis the commoner clinical syndromes.

The almost meaningless titles of "hypopituitarism" and "hyperpituitarism" are rapidly disappearing from the literature, and I feel confident that in the near future the confusing mass of proper names with which the clinical syndromes of pituitary dysfunction have been baptized, will be discarded and that the various disturbances will be classified according to the specific hormone or hormones involved in their production. Realizing that such a classification is at present beyond my reach, I have described the clinical syndromes under their accepted text book titles and have merely attempted to suggest the possibilities of the more satisfactory classification which further knowledge may bring.

Clinical Syndrome.	Growth Factor.	Sex Factors.	Metabolic Factor.	Comments.
Normal	+	+	+	
Fröhlichism	+	−	−	? Also neurohypophyseal disturbance.
Fröhlichism + gigantism	++	−	−	? Also neurohypophyseal disturbance.
Fröhlichism + dwarfism	−	−	−	? Also neurohypophyseal disturbance.
Certain forms of Brissaud's infantilism.	−	−	−	? Some disturbance of thyreotropic factor or of thyreoid gland (hypothyreoidism).
Burnier's syndrome ..	−	−	−	Accompanied by definite evidence of cerebral tumour.
Lawrence-Moon-Biedl (that is Fröhlichism + *retinitis pigmentosa* + cerebral defect).	+	−	−	Possible disturbance of adrenalotropic factor.
Levi-Lorain's infantilism.	−	−	+	
Levi-Lorain's infantilism + progeria.	−	−	+ (? disturbed)	Possible disturbance of adrenalotropic factors or adrenal cortex.
Hypophyseal cachexia +? infantilism (Simmond's disease of childhood).	+ or −	−	−	Possible disturbance of adrenalotropic factors or adrenal cortex. ? Rare or unknown in infancy or early childhood.
Gigantism	++	+	+	(a) "Precocious gigantism". (b) True or permanent gigantism.
Gigantism + obesity ..	++	+	−	
Gigantism + Fröhlichism (? Neurath-Cushing type of infantilism).	++	−	−	
Acromegaly	++	+	+	Extremely rare in childhood.
Hypophyseal hypergonadism.	+	++	+	Sometimes accompanied by adiposity.

The Fœtus and the New-Born Infant.

Probably the growth of the fœtus is controlled to some degree by the activity of the maternal adenohypophysis. The gestational activity and hypertrophy of this lobe are often evidenced by the "acromegaloid" features of the mother during and even for some time after her pregnancy. It seems feasible to suggest that such disturbances of the new-born as mastitis and lactation, swelling of the testes, and even "menstruation" may result from the reaction of the fœtus or infant to the maternal adenohypophyseal hormones.

Levi-Lorain Infantilism.

Modern literature on infantilism may be said to begin in 1871 with a note appended by Lorain to the doctoral thesis of one of his pupils. In this note he suggested the title of "infantilism" for a condition of small stature, slender frame and sexual immaturity. The part played by the pituitary gland did not suggest itself until 1908, when Ettore Levi described a typical case of Lorain's infantilism with evidence of tumour formation in the pituitary neighbourhood. Although Levi believed the pituitary lesion to be a complication and not the cause of the infantilism, it was his careful and complete discussion of the subject which rapidly stimulated the theory that pituitary disturbances may cause developmental arrest.

This so-called Levi-Lorain form of infantilism is probably produced by a depression of the sex and growth factors of the anterior lobe, the metabolic factors usually being undisturbed. Whether this disturbance results from a pure hypoplasia of the acidophile and basophile cells or from tumour formation or pressure cannot be stated definitely, as few established cases have been examined *post mortem*, but, from a review of the literature, it seems that clinical evidence of tumour formation is a rare finding.

The following case is by no means typical of the Levi-Lorain syndrome, but affords an excellent example of well marked depression of the growth and sex factors and possibly some disturbances of other hypophyseal factors, as a result of cerebral tumour formation.

CASE I.—C.R. (see Figure I), a male, aged sixteen years, was reported to have suffered from severe headaches and vomiting between three and four years of age. These symptoms were followed by a gradual failure of vision, of growth and of sexual development. When examined at sixteen years of age he was suffering from bilateral optic atrophy, dwarfism, sexual infantilism, complete absence of any secondary sexual characteristics, an abnormally low systolic blood pressure and some signs suggesting hypothyreoidism. His carbohydrate tolerance was increased and X ray examination of his skull revealed evidence of a large suprasellar calcified cyst (? craniopharyngioma). (Dr. Richmond Jeremy's case.)

FIGURE I.

C.R., aged sixteen years (normal control aged twelve years). Adenohypophyseal dwarfism and sexual infantilism resulting from cerebral tumour formation. ? craniopharyngioma. (Dr. Richmond Jeremy's case.)

The outstanding features of the Levi-Lorain syndrome are dwarfism (with retention of the infantile body and limb proportions), genital hypoplasia and, at a later age, absence of the secondary sexual characteristics. The resultant picture is one of general arrest of the growth and development of the body and all its organs.

CASE II.—R.H. (see Figure II) exhibits the classical features of this syndrome. His dwarfism and sexual infantilism are probably the result of a simple hypoplasia of the acidophile and basophile cells, as no evidence of cerebral tumour formation can be detected. He weighed 4·05 kilograms (nine pounds) at birth and 9·45 kilograms (twenty-one pounds) at twelve months, and his failure of growth and of sexual development was not noticed until he was four years of age. He is now, at seven years of age, only 87·5 centimetres (thirty-five inches) in height (that is, the approximate height of a child of three years) and weighs 11·6 kilograms (twenty-eight pounds) (that is, the approximate weight of a child of two years). Apart from other characteristic features, he exhibits well marked hypogonadism and infantile body proportions. There is evidence of some disturbance of his carbohydrate metabolism, his osseous age is approximately that of a child of three or four years, and his *sella turcica* is regarded as normal in size and shape.

FIGURE II.

R.H.: classical example of Levi-Lorain syndrome. Aged seven years (normal control of same age.)

At birth these children are usually of normal weight, height and appearance. After a variable period, the duration of which rarely exceeds six years, it is noticed that the child is "small for his age". I have personally observed five children suffering from this disturbance. In four of these the condition was discovered between three and six years of age in the course of clinical examination and had not attracted the parents' attention to any degree, while in one child the condition was evident at about ten months of age. This last child was under my observation from three months of age and it was the failure of his height and weight to follow the expected curves after the age of seven months which led to the finding of other features

suggesting the Levi-Lorain syndrome (see Figures III and IV).

FIGURE III.

R.S.: Levi-Lorain syndrome. Aged three and a half years (normal control of same age). Height 80 centimetres (thirty-two inches), weight 13·05 kilograms (29 pounds). The maturity of his facial expression, his stance and certain other features suggest the possibility of progeria. An intense dark brown pigmentation of any portions of his body exposed to sunlight was noted during infancy. Skiagram of *sella turcica* normal. Glucose tolerance test, normal curve. His osseous age corresponded with that of a child of three years.

Owing to the lack of stimulation from the growth factor, although the child's epiphyseal lines remain open unduly late, his skeleton retains its infantile

FIGURE IV.

Another view of child shown in Figure III.

height and proportions. The total skeletal height is far below normal and the measurement from the *symphysis pubis* to the vertex remains greater than that from the symphysis to the soles of the feet. The bones are small and delicate and the centres of ossification are late in appearing. I have been particularly surprised to note that in three of the small group of five children under my care the eruption of the primary dentition and even of the first molars has been obviously less delayed than the rest of the child's physical growth.

There is rarely any evidence of mental retardation, and to the lay observer the picture presented by the child during his early years is usually a pleasant and attractive one. In many cases the graceful, if infantile, proportions of body and limbs, the delicate, even features, possibly a "peaches and cream" complexion, fine, silken hair, and a mental alertness far ahead of the child's apparent age naturally prove pleasant antidotes to

FIGURE V.

B.E.: Levi-Lorain syndrome. Aged seven years (normal control of same age). Height 90 centimetres (36 inches), weight 15·3 kilograms (34 pounds). Skiagram of *sella turcica* normal. Osseous age approximately that of a child of four to five years. Increased carbohydrate tolerance.

any misgivings about his growth which the parents may experience. It is the presence of these attractive features which often makes it difficult to persuade parents that anything is amiss, until the obvious dwarfism and sexual infantilism of later years are apparent.

As age progresses, the clinical picture becomes more striking. The voice retains its high-pitched, childish character, the protuberant abdomen and low umbilicus of infancy persist, the genital apparatus remains in an infantile state (the testes being frequently undescended) and there is an obvious absence of secondary sexual characteristics. Many exhibit a surprisingly mature expression,

which is by no means compatible with even their chronological age and which sometimes progresses to the wizened, aged appearance of progeria (Hastings Gilford[2]). It seems that this prematurely senile appearance may be the result of some disturbance of the adrenalotropic factor or possibly of the adrenal cortex itself (see Figures III, IV and VI).

FIGURE VI.
R.K.: Levi-Lorain syndrome. Aged eight years (normal control of same age). Surprising maturity of facial expression. X ray appearances of *sella turcica* normal. Increased carbohydrate tolerance.

Before a diagnosis of Levi-Lorain's infantilism is assumed, the statural heights of the family should be reviewed, the child's dietary and general physical condition should be carefully considered, and the other known causes of arrested physical development should be excluded as far as possible.

The history of neonatal dwarfism and the normal sexual development of the primordial (or ateleliotic) dwarf should prevent confusion with this type. Thyreoid, cœliac, pancreatic and renal dwarfism and the stunting of growth which accompanies certain bone dystrophies and which may accompany congenital heart disease, congenital syphilis and other chronic infections, should be readily differentiated by appropriate investigations.

Babinski-Fröhlich Syndrome or Dystrophia Adiposo-Genitalis.

In 1900, while Fröhlich and Cushing were working in Liverpool, Babinski recognized and published the first case of *dystrophia adiposo-genitalis*. During the next year, in Vienna, Fröhlich, who was unaware of Babinski's report, published the full details of a similar case, which excited much attention and resulted in his name being associated with the syndrome. The fact that both of these original patients were found to be suffering from chromophobe adenomata may possibly explain the undue prominence which is still given by many text books to cerebral tumour as a causal factor in Fröhlichism.

The classical features of this dystrophy are obesity (which usually exhibits a "feminine distribution") and genital hypoplasia, with or without some disturbance of skeletal growth.

The exact status of the disturbance remains uncertain, as clear-cut evidence of its hypophyseal or cerebral origin is still lacking. But, if we assume that adenohypophyseal dysfunction is responsible, we may regard the sexual infantilism and the obesity as resulting from depression of the gonadotropic and metabolic factors. The growth factor may be disturbed in some cases and apparent specific disturbances of the adrenalotropic or thyreotropic factors may sometimes be suggested. Frequently the disturbance seems to be a temporary one, the affected children losing most or all of their obesity and attaining normal sexual development at a later date.

FIGURE VII.
R.I.: possibly an example of the Levi-Lorain syndrome. Aged five years. Depression of growth factor. Height 82·5 centimetres (33 inches), weight 15·3 kilograms (34 pounds). Some depression of the gonadotropic factors (relatively small penis and both testes undescended) and possibly some disturbance of the adrenalotropic factor or adrenals (growth of dark hair on cheeks, as far as the angle of the mandible, and profuse growth of long dark hair on forearms). X ray examination of *sella turcica* normal. Glucose tolerance test, normal curve. Wassermann reaction negative.

Typical examples of Fröhlichism have been reported as occurring after various forms of encephalitis, including chorea (if this may be regarded as such), after meningitis, after chronic hydrocephalus, after long illnesses such as typhoid fever, and as a result of cerebral tumour formation. Apparently in all these conditions mechanical or inflammatory injury of the hypophyseal mechanism is responsible for the production of the syndrome. It is probably the exception rather than the rule for Fröhlichism to result from cerebral neoplasm in childhood (*vide infra*).

CASE III.—R.H., a healthy boy of normal weight and proportions, was admitted to the Royal Alexandra Hospital for Children at seven years of age, suffering from a meningococcal meningitis. Two years later he evidenced gross Fröhlichism and some gigantism, while seven years after his illness, at the age of fifteen years, he was again of approximately normal proportions, weight and sexual development. (Dr. Edgar Stephen's case.)

CASE IV.—J.L., a girl, attending Dr. Willcocks's outpatient department at the Royal Alexandra Hospital for Children, suffered her first attack of chorea at six years of age. Her parents state that her weight was then 20·7 kilograms (forty-six pounds) (approximately normal for her age) and that she seemed of normal height. When seen at eight years of age, she was 140 centimetres (fifty-six inches in height) (that is, nearly 22·5 centimetres or nine inches over height for her age) and weighed 36·9 kilograms (eighty-two pounds) (that is, nearly 13·5 kilograms or thirty pounds over weight for her age).

According to her parents, the rapid increase in weight and height began about three months after her attack of chorea.

I realize that the typical clinical picture presented by this syndrome is well known to all of you, but I propose to refer briefly to certain features which may be of interest. As a rule there is little or no obvious disturbance of skeletal growth, but occasionally gigantism and, more rarely, dwarfism (possibly the so-called Brissaud's infantilism) may be exhibited. Usually the excess of subcutaneous fat is particularly emphasized over the hips, buttocks, upper portions of the thighs, abdominal wall, breasts and perhaps shoulder girdles. But in many of the younger children I have noticed that the distal portions of the extremities share in the adiposity and that the distribution of fat is similar to that of the overwell-nourished infant. I think that the definite evidence of hypogonadism afforded by the diminutive external genitalia and the obviously abnormal distribution of the fat in boys may possibly explain the frequent statement that the condition is more commonly found in the male. The skin is usually described as pale, cool and soft, but may be dry and harsh in those cases in which the thyreoid or thyreotropic factor appears to be disturbed. In the older children hypotrichosis of the trunk and limbs is evident. Many of the mothers remark that their children bruise very easily and that they seem to perspire surprisingly little for children of their obese proportions. The "alabaster-like" colouring and also the gross freckling of exposed parts which sometimes occur, possibly result from associated disturbance of the adrenals or the adrenalotropic factor.

The majority appear to be of normal intelligence, while a small proportion may suggest minor degrees of mental retardation. In those rare cases of Fröhlichism which are accompanied by *retinitis pigmentosa* (that is, the Lawrence-Moon-Biedl syndrome) mental defect is a prominent and almost constant symptom. This Lawrence-Moon-Biedl syndrome is usually familial and is frequently accompanied by polydactylism or syndactylism.

CASE V.—K.G. (see Figure VIII), a male of ten years, attending Dr. Laurence Hughes's out-patient department at the Royal Alexandra Hospital for Children, exhibits gross, almost confluent freckling of all exposed parts and some increased deposition of fat over the buttocks and lower limbs, while the face, upper limbs and upper portion of the trunk remain lean. His genital development is retarded, he suffers from *retinitis pigmentosa*, and has an increased tolerance for carbohydrates.

I have included the above case because it seems to be closely allied to the Lawrence-Moon-Biedl syndrome, which is rarely seen in its entirety.

There seems to be a tendency amongst some observers to apply the title of Fröhlichism to any child whose genital development seems to be slightly delayed and who exhibits more adiposity than its fellows. In many such children the title is barely justified, unless some further suggestion of hypophyseal or diencephalic disturbance is revealed. A careful review of the diet should be of some help in attempting to exclude cases of the simple exogenous obesity of childhood. If the mothers' statements may be accepted as accurate, it seems that the majority of children suffering from Fröhlichism are surprisingly small eaters. This has seemed to me to be a striking feature of many of their histories and affords an interesting contrast to the ravenous appetite attributed to acromegalics.

Occasionally associated with Fröhlichism are various symptoms and signs, such as polydipsia, polyuria, disturbances of respiration and heat regulation, undue somnolence and "sham rage" attacks, which are not adenohypophyseal in origin and which have therefore been excluded from this discussion. For the same reason discussion of the part played by the neurohypophyseal mechanism in the production of this form of obesity has been avoided.

FIGURE VIII.
K.G., aged ten years: *retinitis pigmentosa*, some hypogonadism, slight lipodystrophia, gross freckling of exposed parts of body. ? Lawrence-Moon-Biedl syndrome. (Dr. Laurence Hughes's case.)

Hypophyseal Cachexia.

So much emphasis has been laid on adiposity that the fact that there are cachectic as well as fat types of dyspituitarism has not received the attention which it deserves. A partial or complete destruction of the adenohypophysis, following some severe generalized infection, has been described by Simmonds as resulting in a condition of hypophyseal cachexia, which may be accompanied by some degree of infantilism. I have been unable to find any reference to an established case of Simmond's disease occurring before adolescence. Cameron has made the helpful suggestion that Simmond's disease may be regarded as bearing the same relation to the Levi-Lorain syndrome as myxœdema does to cretinism.

I think that it is reasonable to suggest that the extreme wasting which is sometimes seen in children suffering from slow-growing cerebral tumours, chronic meningitis and hydrocephalus may be regarded as partly hypophyseal in origin. Despite a relatively satisfactory caloric intake and little or no vomiting, these children frequently exhibit an extreme degree of emaciation. Possibly the extreme cachexia which sometimes accompanies post-encephalitic Parkinsonism in children may be placed in this same category.

Hypergonadism.

A simple hypergonadism, apparently of hypophyseal origin, is occasionally recorded in childhood. The following case, although not primarily hypophyseal in origin, possibly resulted from an irritative lesion of this mechanism.

CASE VI.—J.N. (see Figure IX) developed pubic hair at three years and at four years of age began to suffer from "sham rage" attacks. At eight years of age he exhibited the sexual development and secondary sexual characteristics of late adolescence. Autopsy revealed an astrocytoma of the floor of the third ventricle. (Dr. Wilfred Vickers's case.)

FIGURE IX.
J.N.: hypophyseal hypergonadism. Aged eight years. (Dr. Wilfred Vickers's case.)

CASE VII.—P.T. (see Figure X), a girl of five years, might also be regarded as furnishing an example of hypophyseal hypergonadism. Her breast development became obvious at three years. At five years of age she now exhibits scanty pubic hair and fully developed breasts. There has been no sign of menstruation and no evidence of an intracranial, suprarenal or ovarian tumour can be detected.

References to an adiposo-hypergenital syndrome may be found in the French literature,[3][4] the main features being florid obesity, precocious puberty, occasionally a tendency to hirsutes, and in many cases a vivacity of movement which is in striking contrast to the heaviness of the figure.

The following case would seem to be a possible mutation of this adiposo-hypergenital syndrome.

CASE VIII.—H.G. (see Figures XI and XII), aged sixteen years, one of twin girls, has been attending Dr. Laurence Hughes's out-patient department since she was eleven years of age. I am grateful to Dr. Hughes for the following facts about this patient:

At seven years of age the patient was noticed to be gaining weight much more rapidly than her twin sister. At eleven years menstruation commenced, she exhibited fully developed external genitalia of the adult type and a marked degree of adiposity, which had a distribution similar to that seen in a middle-aged woman.

Her lower jaw is large and prominent, and her teeth are "spaced" in the upper jaw. Her speech and intelligence are poor for her age. Her basal metabolic rate was found to be lower than normal. Her tolerance for glucose is increased. An X ray examination of her skull did not reveal any abnormality, and her visual fields are apparently normal.

It is questionable whether a classical case of Cushing's pituitary basophilism has ever been observed in children, and it is improbable that true acromegaly ever occurs before adolescence.

FIGURE X.
P.T., aged five years. ? hypophyseal hypergonadism.

Gigantism.

During infancy and childhood hyperfunction of the acidophile cells and consequent exaggeration of the growth factor may produce various types of somatic overgrowth or gigantism. The forms of adenohypophyseal gigantism which are evidenced before puberty may be classified into three groups:

1. *The Gigantism of Infancy.* The gigantism of infancy may be recognized even before four months, but rarely becomes obvious until nine months of age or later. The sensational gains in height and weight which some of these infants achieve may suggest gross over-feeding, until the underlying endocrine disturbance is realized. Probably some of these infants suffer from Fröhlichism *plus* gigantism, while others seem to evidence pure gigantism.

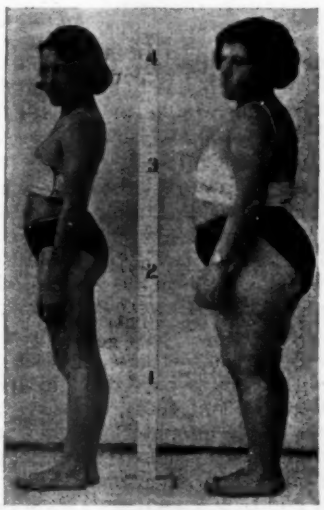

FIGURE XI.
H.G., aged sixteen years, photographed with her twin sister. Possibly an example of the hypophyseal adiposo-hypergenital syndrome. (Dr. Laurence Hughes's case.)

CASE IX.—C.C. weighed 3·6 kilograms (eight pounds) at birth and during nine months of breast feeding gained

weight with abnormal rapidity, attaining a weight of 15·3 kilograms (thirty-four pounds) at ten months of age. At eighteen months of age he was 86·25 centimetres (thirty-four and a half inches) in height (normal approximately 75·0 centimetres or thirty inches), weighed 17·1 kilograms (thirty-eight pounds) (normal approximately 11·7 kilograms or twenty-six pounds), and proved an easy winner in a baby competition. At this age his hands were as large as those of a child of three or four years; his skin was dry and somewhat scaly, his carbohydrate tolerance was slightly increased, and X ray examination revealed an abnormally small *sella turcica* and also an area of subcutaneous calcinosis in the neighbourhood of his left elbow joint. His parents were of approximately normal height and weight, and a review of his diet did not suggest over-feeding.

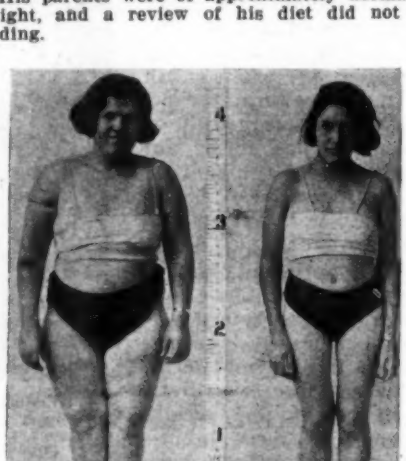

FIGURE XII.
Another view of the child shown in Figure XI.

It is difficult to correlate the subcutaneous calcinosis with any particular hypophyseal disturbance, but it is interesting to note that such deposits have been reported in cases of progeria.

2. *Precocious Gigantism.* In my experience, marked precocious overgrowth of the whole body is not uncommon in children approaching puberty. This modified form of gigantism is usually transitory and the majority of these children do not appear to be much above the average size in later years.

3. *True Gigantism.* True gigantism, that is, permanent gigantism, with heights of 195 to 240 centimetres (six and a half to eight feet) or even of 300 centimetres (ten feet), is a rare disturbance of childhood. Behrens and Barr have reported the case of a boy who weighed 4·0 kilograms (nine pounds) at birth, 13·5 kilograms (thirty pounds) at six months, and 27·9 kilograms (sixty-two pounds) at eighteen months of age. At nine years of age he measured 182·5 centimetres (six feet one inch), weighed 80·1 kilograms (twelve stone ten pounds) and was able to pick his father up and carry him about. His *sella turcica* was abnormally large and there was a shadow suggestive of a craniopharyngioma.

It is very doubtful whether "partial gigantism" or hemihypertrophy may be regarded as being of adenohypophyseal origin.

The gross enlargement of the sebaceous ducts and the thickened flaccid state of the skin so commonly seen in gigantism and in many cases of acromegaly contrast very sharply with the fine texture and the firmness of the skin which are characteristic of both the Levi-Lorain and Fröhlich's syndromes. This contrast in the condition of the skin and the fact that the usual course and history of *acne vulgaris* supports the theory of an endocrine ætiology suggest that the adenohypophysis may play some part

FIGURE XIII.
B.W., aged eight years: dwarfism, obesity, polyuria and polydipsia. Fundi and fields of vision normal. X ray appearances of skull normal. No reaction to the Wassermann test. Increased carbohydrate tolerance. During the past six months there has been a normal increase in growth, a marked diminution in the polyuria and a decrease in the polydipsia and the obesity.

in predisposing children and adolescents to this skin disorder. As a further evidence in favour of this hypothesis Dr. Hansman has informed me that he has known pituitary extracts to be of definite value in the treatment of *acne vulgaris*. It will be interesting to note the response of this skin lesion to injections of satisfactory adenohypophyseal extracts, when such preparations are available.

Diagnosis.

The diagnosis of pituitary dystrophies in childhood seems to depend in the main on clinical examination and the exclusion of other causal factors, for both radiographic and biochemical examination are frequently of little value. Radiographic evidence of changes in the *sella turcica* and its neighbourhood may be found in apparently normal children and are seldom of any diagnostic value unless there are gross alterations in size and configuration of the fossa, or definite signs of calcification in the neigh-

bourhood. The fact that a *sella turcica* appears normal does not negative a diagnosis of pituitary disturbance, nor does a report of some change in the size and shape of the fossa establish such a diagnosis. I have reviewed the X ray reports of the skulls of twenty-two children who were apparently suffering from various pituitary dystrophies, and have found that gross changes in the *sella turcica* or in its neighbourhood were reported in only three cases.

In my opinion, the difficulty of assessing with any degree of accuracy the osseous age of a child makes such an investigation of doubtful value before adolescence. The results of glucose tolerance tests of these children are extremely variable. The generally accepted statement that they usually exhibit a high carbohydrate tolerance is borne out by a certain number of cases, but it is by no means a constant finding. Reviewing the results of glucose tolerance tests carried out on twenty-eight children apparently suffering from pituitary dysfunction, it was found that approximately 60% showed some increase in carbohydrate tolerance, in over 30% the curves were apparently normal, while in the remaining cases surprisingly divergent results were obtained which were difficult to interpret. The obvious difficulties associated with the accurate estimation of the basal metabolic rate in children and the absence of any satisfactory tables of normal values for these ages both detract from any particular value which might be attributed to such results.

Probably in the near future, when satisfactory and potent extracts of the various adenohypophyseal factors are available, accurate diagnosis and classification will be possible, being based upon the patient's response to injections of the appropriate hormone.

Despite the statements to the contrary which still appear in the literature, I should like to suggest that the great majority of cases of dyspituitarism in childhood are not the result of tumour formation. The following evidence may add some support to this suggestion:

1. Englebach[6] found *post mortem* evidence of tumour formation in only 4% of 375 pituitary disorders.

2. Cushing,[7] in demonstrating eighteen intracranial tumours in children, emphasized to his audience the rarity of dyspituitarism in such cases and pointed out that only one of these children showed any such changes.

3. A review of the clinical histories of a series of twenty-four children suffering from cerebral tumours (taken from the records of the Royal Alexandra Hospital for Children) revealed only one case which suggested pituitary dysfunction—that of J.N., who had an astrocytoma of the floor of the third ventricle.

4. I have investigated and have observed, over periods varying from six months to six years, a group of more than twenty children apparently suffering from pituitary disturbances. In only one of these children (L.R., possibly suffering from a craniopharyngioma) was there any evidence of cerebral tumour.

Treatment.

Any systematic discussion of treatment is far beyond the scope of this short paper, but the following brief comments on certain therapeutic aspects may be of some interest.

It is recognized by most authorities that the various whole gland and other pituitary preparations for oral administration are probably valueless and that those injectable extracts of the various adenohypophyseal factors which are at present available possess little or no therapeutic value. Talbot[8] and others state that the present injectable proprietary preparations are practically inactive in the human, but Englebach has reported definite improvement in the rate of growth of a girl suffering from the Levi-Lorain syndrome. Englebach[9] treated this child with an extract of the adenohypophyseal growth factor, giving increasing doses of daily, intradermal injections.

I have treated two patients suffering from the Levi-Lorain syndrome with injections of an extract of the adenohypophyseal gonadotropic factor (kindly donated by Parke, Davis and Company). The injections were given biweekly for approximately five months, but neither of these children showed any obvious change in their sexual development or any increase in their rate of growth.

The obesity of Fröhlichism does not appear to yield to any known proprietary preparation, but some cases may respond to dietetic restrictions, and thyreoid extract may be of value in those cases in which there appears to be an obvious disturbance of the thyreotropic factor. Therapeutic application of X rays to the gland has been used with some success in the treatment of adenohypophyseal gigantism and may provide a rational form of treatment in the most severe forms of this disease.

Potent active extracts of the adrenal cortex have been available to clinicians for some years, but cases suggesting Addison's disease seem to be almost unknown in children under twelve years of age. The discovery of definite indications for the use of extracts of adrenal cortex during this age period would result in a brilliant addition to pædiatric therapeutics. As regards the hypophyseal disturbances of childhood, the above conditions are exactly reversed—the various syndromes are well known to clinicians, but laboratory workers have not yet produced those satisfactory therapeutic extracts which almost certainly will be available in the near future.

Acknowledgements.

In conclusion I wish to thank those of my colleagues who have allowed me to examine and report their cases, and Dr. Phyllis Anderson for carrying out and tabulating a number of glucose tolerance tests.

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EPILEPSY FROM A PÆDIATRIC POINT OF VIEW.

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EPILEPSY has always furnished a happy hunting ground for theorists. It can hardly be otherwise. But most authorities see and study the condition in its well established adult stage. In children disease processes are seen in their simpler forms, and I believe it is only by the study of epilepsy in early life and in its relation to development of the function of the nervous system that we can arrive at any clear conception of epileptic fits and their variants. Gross pathological changes found at autopsy in epileptic dements can never explain epileptic phenomena. Epileptic fits or their equivalents are merely symptoms of some pathological condition, but the pathological conditions that cause them are innumerable. Nevertheless I do not think it helps understanding to speak of them as "the epilepsies". I believe there is but one pathological condition as the basis of epilepsy, a developmental or less often acquired and often temporary disturbance or deficiency of neuronic associations, other conditions being but exciting causes. That only, I think, can explain the convulsions that can be produced under certain conditions in healthy persons, as well as the fits of idiopathic epilepsy.

A study of my cases and records over the past thirty years has convinced me that the only possible chance of determining causes is to trace the patient back to early infancy, even to prenatal life, and that the prevention of epilepsy is closely bound up with the care of the infant. Even with the greatest care there will always be some children doomed to epilepsy, even if they escape infantile convulsions.

In the first place, as Kinnier Wilson emphasizes, such positive signs as convulsions cannot possibly be due to destruction of motor cells; they must be due either to release of inhibitory control or to irritation of these cells; they may in fact presuppose normality of function of these cells. Hence, when Aldren Turner found some failure of development as well as degeneration of motor cortical cells, he might also have found similar failure in the upper cortical layers or in the cells subserving the distribution and "damping" of sensory impulses, the cells of transcortical association. Again, Kinnier Wilson emphasizes that lesions are simply tissue reactions to toxic or traumatic damage and that disturbance of function may easily occur long before and even without the occurrence of a definable lesion, and further, that the manifestations in the form of symptoms may arise at a point far distant from the original place of disturbance. It may not be necessary to go so far as Foerster and others have gone and postulate an efferent sensory system inhibiting the afferent sensory system. I prefer to think of the distribution and regulation of sensory impulses as a function of the synaptic relations of the brain cells as a whole. This carries us back to the old ideas of Gowers and others, ascribing epilepsy, as well as chorea and tics, to disturbances of synaptic relations either on the motor or on the sensory side. Then the finding of Turner as regards the under-developed brain cells regains some significance, particularly if we appreciate the fact that at the time he wrote the subdivisions of the cortical layers and the present conceptions of their functions were not realized. I think too much has been made of the old conception of Jackson of a "discharging lesion" from which radiation occurs; the obvious positive symptoms, such as convulsions, have tended to overshadow the very numerous negative signs of disturbance that are found in epilepsy. It must be remembered that removal of the higher levels of cerebral function makes positive motor activities more readily elicited than under normal conditions. Yet there are epileptic conditions that have their origin in the highest brain levels, especially those displaying psychical manifestations, either as an aura or throughout the attack. Cerebral disturbances in infancy and childhood are clearly expressed by the state of the nervous system as regards anatomical and functional development. There are three main anatomico-physiological factors to be kept in mind in their relation to epilepsy:

1. Although at birth a child possesses almost all the neurones it will ever possess, yet very few are functionally active in that their axones are not yet completely medullated.

2. The check to effector response, which we know as inhibition, is therefore still poorly developed.

3. There is very rapid growth of the brain, with rapid and irregular medullation of neuraxones and irregularly increasing functional activity of the psycho-associational neurones.

It is therefore the association and coordination of cerebral functions that are important; the more complicated the required action and the greater the urgency and rapidity necessary for its performance, the more likely are disorder and, therefore, convulsion or other disturbance to occur, the particular clinical manifestations depending upon the area the

functions of which are disturbed. It may be noted that tonicity is more common the younger the child and the more retarded the cerebral development, while in adults it is likely to indicate disturbance or dissociation of cerebello-ponto-striate functions.

That fits and their equivalents may be produced in apparently normal people by toxins or poisons, or even by emotional shocks of sufficient intensity, is well recognized and we are justified in a diagnosis of epilepsy only in the absence of an adequate cause. Absinthe, continued alcoholic excess, and uræmia all cause fits, and Collier has postulated a metabolic dyscrasia as a sufficient cause of epilepsy, but metabolic disturbances in so many directions have been observed in epilepsy that it is difficult to believe that they play any great part in ætiology, except as possible excitants of particular attacks in an otherwise susceptible person. Similarly many gross pathological lesions have been found in epilepsy, but it is probable that they act, not as stimulants to the cortical motor cells, but rather through interference with inhibition or cortical association, thus lowering the threshold of epilepsy.

The clinical aspects of epilepsy are explicable only if we can conceive a condition of neuronic insufficiency, either developmental, arising from some influence either antenatal or in the early years of infancy, or else some lesion which by its influence reproduces an infantile type of cerebral reaction. For we can confidently assume that if normal brain development occurs within the first fifteen months after birth there is little likelihood of developmental defect later; but, as Collier has pointed out, gross cerebral lesions or severe toxic conditions can reproduce a state anatomically and physiologically closely akin to that arising from faulty development. Hence the vast majority of patients with idiopathic epilepsy have been epileptic from infancy, although the first manifestations may have been delayed for many years.

It is impossible to distinguish the convulsions of infancy from those of epilepsy; they differ in symptoms only on account of the age, and in prognosis only in so far as the brain in infancy is still in the developmental stage, and may, with proper management, proceed to full and normal development. In comparatively few cases do the convulsions of infancy pass directly into epilepsy in later life, so that we may say that an infant under two years of age who has had convulsions is not likely, *ipso facto*, to become epileptic, but when a child over the age of two years has even one convulsion it must be regarded, or at least suspected, as indicative of latent epilepsy. Many unrecognized attacks of *petit mal* or of epileptic equivalents occur in infancy and are often precursors of major epilepsy.

Causes of Neuronic Insufficiency.

Analysing our clinical records in a search for factors that might explain faults in neuronic development, we have no difficulty in finding many potent influences.

Heredity as a factor has been much misunderstood. Epilepsy is not often directly inherited, although there may in some cases be inherited a failure in neuronic development, but this may manifest itself in many other ways. But, as is to be expected, there are some families in which the frequent occurrence of epilepsy cannot be explained except by heredity:

K.S., aged nine and a half years, has had fits for three months without definite exciting cause. He was a breast-fed baby; no convulsions occurred in infancy. His maternal grandmother is epileptic; the mother was epileptic from nine to eighteen years of age; his sister had two fits at three, and his brother two fits at the ages of three and seven years.

Mental defect is present in many cases, but is rather the result of a widespread neuronic deficiency and an accompaniment of epilepsy than the cause, but it may follow the vascular disturbances and degenerations of confirmed epilepsy.

Congenital syphilis is a definite factor in a few cases, but statistics show that the Wassermann reaction is not much more frequently positive among epileptics than among non-epileptics. Nevertheless there are families in whom the Wassermann-positive children are epileptic and the Wassermann-negative are free. Syphilis is probably in these cases the deciding factor.

Debilitating diseases of the parents or toxic conditions, such as chronic alcoholism or plumbism, are factors in some cases. Nutritional disturbances, generally grouped under the term marasmus, are so often voluntarily tendered by parents as an explanation that it is impossible to ignore the great importance of dietetic factors and of nutritional disturbances as causes of neuronic insufficiency. There can be no doubt that cerebral development is greatly influenced by nutrition and that in pronounced cases of rickets it is apt to be grossly defective; hence some Continental authorities have considered rickets an essential factor in the causation of epilepsy. The spasmophilia of infancy, which is regarded as rhachitic, is not far removed from epilepsy. With our present knowledge of nutrition we may readily accept the view that dietetic faults may exert such a powerful effect upon neuronic development, especially during the most important first fifteen months of life, that the child becomes and will remain throughout life a potential epileptic awaiting an adequate exciting cause of fits. Gross cerebral lesions, especially those impinging on the sensori-motor cortex or those leading to cerebral changes over a wide area, may not only so interfere with function as to be an effective cause of epilepsy, but may also prevent normal development; this is illustrated by the conditions of cerebral diplegia in which fits are so common. Severe toxic conditions may lead to destruction of neurones, as also may inflammations, such as encephalitis and meningitis, and degenerations of any origin are productive of a condition akin to failure of development.

To sum up, while I think it unwise to rely on statistics unless large numbers of cases under all conditions are dealt with, yet my records show that approximately 5% of epileptics exhibit some

hereditary factor, 5% of fits have definitely followed trauma, either birth injury or concussion, 3% have occurred during or shortly after severe infections, particularly diphtheria, 8% of epileptics have been congenitally mentally defective, and 14% have given a history of grossly defective feeding in infancy, many of them showing well marked rhachitic signs. Congenital syphilis has probably accounted for 4%, this being within the limits found in this country in a hospital out-patient children's department among all patients applying for treatment; a Wassermann test has not been performed in all cases, so that syphilis may be partly responsible for a larger number. In 20% no definite cause was found; 14% followed convulsions in infancy.

Exciting Causes of Fits.

The causes that excite individual fits or their equivalents are numerous, and it seems that one fit predisposes to other similar fits, yet, after daily fits after a period of years, there may be a sudden cessation. No satisfactory explanation has so far been found for that phenomenon.

Psychical causes are responsible for many cases:

E.W., aged seven years, recently in hospital with diphtheria, was taken to see his dead father. He took a fit at the bedside and became a confirmed epileptic.

A.M., aged two and a half years, was forced to go close to a steam-roller, of which she was frightened, and promptly took the first fit of many.

J.W., aged thirteen years, saw his companion killed by a motor car.

J.B. saw his brother killed in a cycling accident.

In these cases fits followed directly and were frequent afterwards. But in many cases fits have been attributed to mental shock or injury sustained long before the first fit; in most of these other causes will be found and the shock or injury may have no relation to the fits. Fits often occur in children just before they fall asleep; they are comparable with the myoclonic attacks that often occur in healthy adults at that time.

Toxic causes are another large class; with them may be included the convulsions that occur at the onset of febrile disorders in children, as well as those arising during anæsthesia; I have seen two cases under pure chloroform in patients who had no fits before or after. In adults excessive smoking, or chewing of tobacco, or alcohol seems to increase the number of fits. It must be remembered that toxins of sufficient potency, as well as vascular disturbances such as occur in heart-block, will cause fits in otherwise healthy people; similarly, terminal convulsions occur with severe illnesses. It would be absurd to suggest that sufferers from these conditions are epileptics, yet the essential nature of the conditions that result in fits must be the same.

Reflex causes are often regarded, especially by parents, as responsible for fits, but, although in a few cases the fits cease after removal of tonsils and adenoids or after circumcision, in most they return at a later date.

It is difficult sometimes to decide whether a patient or his "turns" are epileptic or not; even loss of consciousness is not always present, even in major attacks. One adult patient with generalized epileptiform fits following hemiplegia was quite aware of all that went on around her on one occasion when she was in a condition of *status epilepticus* for twelve hours, and has always been conscious during subsequent fits. Since Sir William Gowers included sudden starts, certain forms of vertigo, temporary visual defects, and vaso-vagal attacks in the category of epilepsy, the tendency has been to extend the classification further to include, on the motor side, phenomena ranging from myoclonic spasms to the convulsions of major epilepsy and, on the sensory side, sudden transitory disturbances of sight, hearing and sensation, and migraine.

Treatment.

It is sometimes said that we have made no progress in the treatment of epilepsy. It must be admitted that some patients will proceed to a stage of dementia whatever we do, but we certainly study and understand our epileptics better, and I think we can do much more for them now than we could twenty or thirty years ago. In the first place, the epileptic is not regarded as being "possessed of a devil", as in the middle ages, nor even as a hopeless proposition, as was the case thirty years ago. We know that we should interfere as little as possible with his normal life; in most cases it is quite unnecessary to prohibit school attendance, even though it may be necessary to advise his teachers of his condition.

The diet should be regulated in the direction of restriction of carbohydrates, as has been my practice for the past thirty years, following the suggestion of Francis Hare, then of Brisbane. It is sometimes helpful to adopt a definite ketogenic diet.

Bromides are no longer our standby; some patients do well with them, but "Luminal" or some other member of the barbiturate group has gradually usurped their position. Our great object is to stop fits, and we can do that more readily with "Luminal" than with bromides. One dose daily is often sufficient, if given shortly before the time a fit usually occurs.

While I do not subscribe to the psychogenic theory of epilepsy, I do not think sufficient use is made of suggestion in treatment, particularly in those cases that start with an aura of emotional type or that are associated with phobias of different kinds.

As I have suggested, epilepsy can sometimes be prevented by proper feeding in infancy, attention to environment, and avoidance of illnesses and of severe emotional strains during the period of rapid brain development in infancy and childhood. It is necessary, of course, to seek out and remove any conditions likely to prove excitants of attacks or to interfere in any way with the general health.

Summary.

1. There must be one single essential reason why fits should occur in healthy individuals as well as in those affected by gross cerebral lesions.

2. Sir William Gowers's theory of disturbance of neuronic function seems to be the only theory so far propounded that provides such an essential and adequate reason. In other words, the inadequacy of neuronic development or the occurrence of lesions or toxic conditions that reproduce a similar state provides the only satisfactory explanation of epilepsy.

3. The causes generally accepted are but exciting or secondary causes, to which treatment must be directed, but they cannot be the primary cause.

4. Prevention of epilepsy depends upon the minimization or elimination of all influences that lead to deficiency or disturbance of neuronic function.

Reports of Cases.

QUININE AMBLYOPIA.

By E. C. TEMPLE SMITH, F.R.C.S.E.,
Sydney.

I AM prompted to report this case by the letter of Dr. Spring, published in this journal on July 7, 1934. He quotes Fuchs as saying that some cases of quinine amblyopia have been known to occur after a dose of fifteen grains. I have met such a case.

I was asked by Dr. Curtis Elliott on the morning of March 12, 1933, to come out and a see a young woman of about twenty-five years who had become quite suddenly blind. I found a young unmarried business woman in bed, apparently quite blind, with widely dilated, immobile pupils and an air of distress. The fundi and optic disks were normal. Beyond not having felt very well for a short time, she said there was nothing wrong with her and that she had taken no drug. The blindness had shown itself only when she awoke that morning. I retired somewhat nonplussed. Dr. Elliott then told me that he had elicited the fact tnat she had missed her monthly period. That seemed a clue. I returned and asked her how many tablets of quinine she had taken. She replied: "three". I advised rest in bed and vaso-dilators.

I saw her at my rooms on March 30. Her vision with correction was $^6/_6$ and $^6/_{12}$. Her fields, as shown in the appended charts, were very much restricted. The disks were pale and the arteries contracted. I impressed on her the importance of keeping under observation, while giving her a cheering prognosis (with mental reservations). In spite of this I have not seen her since, and Dr. Curtis Elliott has been unable to trace her whereabouts. Her surname begins with L. If any of my colleagues have come across her, I should be very glad to have her present vision and fields, so that one could be guided in prognosis. I can only suppose that, if her story was correct, she had an idiosyncrasy for quinine, as they were only five-grain tablets.

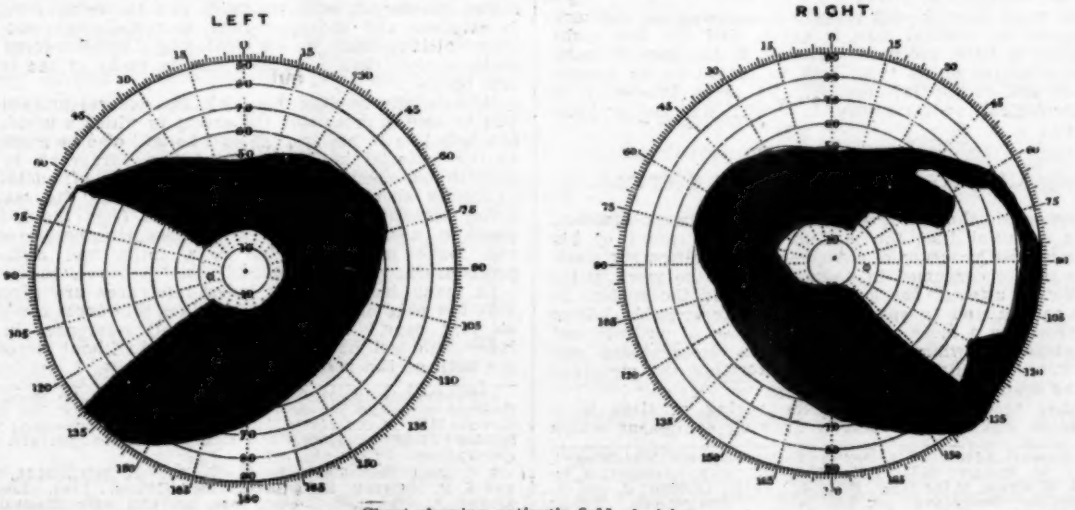

Chart showing patient's field of vision.

LIPODYSTROPHY.

By IAN GRAHAM, M.B.,
Boggabri, New South Wales.

THE case I am reporting is, I believe, so rare that I think it may be of interest to fellow practitioners.

Miss A., aged twenty-four years, was a domestic employee. Both her parents were alive; she had three brothers and three sisters; all were healthy.

The patient was quite healthy until five years ago, when she developed dysmenorrhœa two years after the establishment of menstruation, which did not occur until the age of seventeen years. The menses have occurred only every forty-two days and last from four to five days, the loss being normal.

Four years ago, that is, one year after the incidence of dysmenorrhœa, she noticed that she was losing weight without loss of appetite or other subjective phenomena beyond polyuria.

Three and a half years ago she had an appendicectomy performed as treatment for a recurring pain in the right iliac region. This pain was not relieved.

During the last few months she had noticed a swelling in the neck at the time of her menstrual periods and presented herself for examination with the statement that she was suffering from goitre.

On examination the most outstanding feature was the gross emaciation of the face and hands. The eyes were sunken, the cheeks were so hollow that the zygomatic arches and the mandible and even the alveolar margins of the maxillæ stood out in bold relief, giving the features almost the appearance of a caricature.

The arms, thorax, and the abdomen above the level of the umbilicus were similarly devoid of adipose tissue, while the abdomen below this level and the legs were well padded with fat and of normal appearance.

There were no abnormal features in the various systems, the urine being clear and the blood sugar normal.

I think that the most interesting feature, apart from the rarity of the case, is the apparent association between the dystrophy and the endocrine dysfunction.

Reviews.

THE PHYSIOLOGY OF SEX AND REPRODUCTION.

RESEARCH into the ovarian hormones and the gonadotrophic secretions of the anterior pituitary gland has been very thoroughly made at the Institute of Animal Genetics of Edinburgh. "Recent Advances in Sex and Reproductive Physiology" is a work in which the author, J. M. Robson, has presented a very recent and complete survey of his subject, with an introduction by Professor Crew, Director of the Edinburgh Institute.[1] The publication is apparently sponsored by Crew and thus bears his hallmark.

Robson deals with the vaginal smear test for œstrum, and he describes uterine and ovarian changes during the menstrual cycle; he also discusses the cause and result of menstruation.

The anterior pituitary secretions are described along the lines indicated by Zondek. It is shown that cessation of ovarian activity results in an increase of the secretion of prolan *A*, and this increase may continue indefinitely. It is thought to be due to loss of inhibitory effect of the ovaries.

Methods of assaying prolan *A* and *B* are given, and the author draws attention to Zondek's findings of excessive gonadotrophic hormones in cases of chorioepitheliomia and hydatidiform mole. Muscle tone and reactivity to oxytoxin are dealt with, but at present have little therapeutic value. Follicular and luteal changes are described, and the author shows that hypertrophy of the myometrium and endometrium is due to œstrin, but that glandular proliferation is due to the *corpus luteum* influence. He discusses the possibility of leucorrhœa being of hormonal origin. He considers that excessive amounts of œstrin account for nervous conditions of the premenstrual hypertension type and that *metropathia hæmorrhagica* is associated with increase of œstrin. Further, the author states that some types of amenorrhœa, and even sterility, are due to lack of œstrin.

The controlling factors of parturition are discussed from the hormonic aspect, and also the influence of hormones upon lactation. The concluding chapter concerns the clinical application of the pregnancy tests and their application to abortion and malignancy. In the same chapter the author discusses the therapeutic use of hormones especially in such conditions as sterility, amenorrhœa, dysmenorrhœa, the menopause, *et cetera*.

The work is very well presented, although for ordinary purposes the medical man is apt to find the first eight chapters a little laboured. The book, however, is most comprehensive of its type, both in regard to its presentation and recent information. To those interested in gynæcological endocrinology it might well be a *vade mecum*.

ANATOMY OF THE NERVOUS SYSTEM.

IN working through a course of practical neuro-anatomy, it is essential that the student make a record of his observations by means of sketches and drawings, but much time can be consumed in doing this. In the work under review an attempt has been made to help the student in this respect and to spare him much unprofitable labour by providing a more or less complete set of outlines and unfinished drawings of the various preparations and specimens usually studied in investigating the structure of the brain and spinal cord.

Other teachers accomplish the saving of time in a different way by providing a drawing equipment which enables the student to make his outlines, at least of the microscopical preparations, in the minimum time; but with larger classes, it is obvious that other methods must be adopted. The present author's solution is a very good one and reduces waste of time to a minimum.[1]

Besides this atlas of "blanks", adequate directions are given for the dissection of the brain, and these are given in "assignments", each calculated to provide work for a three-hour laboratory period. Special consideration has been given to the fact that sufficient human material for large classes is sometimes difficult to obtain, and so instructions are given as to how to utilize material to the best advantage.

An admirable feature of the work is the early introduction of physiological and clinical considerations which help to arouse the student's interest and to stimulate his imagination.

The work is not meant to take the place of the number of excellent text books on neuro-anatomy, but serves merely as a guide to practical work, and in this respect achieves conspicuous success. The instructions are clear, concise, and adequate, and enable the student to proceed without the necessity of looking for the help of demonstrators.

The new edition is produced on good paper and the illustrations are therefore much better reproduced than in the last edition. The binding, too, is different, and the loose-leaf method has been abandoned. The "blanks", however, are perforated and can, if necessary, be removed and subsequently, when filled in, be pasted in again.

PATHOLOGY FOR THE CLINICIAN.

IN "Clinical Pathology" Panton and Marrack have aimed at the production of a book which will be of use to the medical student and the general practitioner.[2] In this they have succeeded admirably, and the book should also find its way into the possession of the laboratory worker. The book, which has now reached its third edition, is divided into eight sections under the headings: the blood, parasitology, puncture fluids, the urinary system, the alimentary system, the eye and the skin, the respiratory tract, and histology.

Naturally, as the authors themselves point out, it is difficult to deal in detail with each of these subjects in one volume, yet by omitting all references and avoiding repetitions the authors have been able to give the salient features of practically every examination which a medical practitioner might demand of a laboratory. Where all sections are good, several call for special mention. In the section dealing with blood, in addition to a description of the various anæmias, the changes found in disease, and the methods of examining blood, there is a comprehensive section on the biochemistry of the blood.

The chapter on puncture fluids and the urinary system is excellent and the cytological, bacteriological, and biochemical investigations are treated in a more ordered and lucid manner than is to be found in many of the larger text books.

By carefully reading the book, the general practitioner will be able to determine the extent to which a laboratory can help him in his practice and he will also be guided as to the material he can submit. One paragraph in the chapter on vaccines is especially worthy of quotation: "Vaccines should not be given merely for the sake of doing something. A coccus extracted from the healthy gums or normal fæces of a hopeless rheumatic subject and made into a vaccine is a futile and dishonest procedure."

In many instances the index references are given to only one page of the text and further references are given on that page. The inclusion of all references in the index could well have been accomplished without increasing the bulk of the book.

[1] "Recent Advances in Sex and Reproductive Physiology", by J. M. Robson, M.D., B.Sc., F.R.S.E., with introduction by F. A. E. Crew, M.D., D.Sc., F.R.S.E.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 259, with 47 illustrations. Price: 12s. 6d. net.

[1] "Neuroanatomy: A Guide for the Study of the Form and Internal Structure of the Brain and Spinal Cord", by J. H. Globus, B.S., M.D.; Sixth Edition, revised and enlarged; 1934. London: Baillière, Tindall and Cox. Demy 4to., pp. 256, with illustrations. Price: 16s. net.

[2] "Clinical Pathology", by P. N. Panton, M.A., M.B., B.C., and J. R. Marrack, M.A., M.D.; Third Edition; 1934. London: J. and A. Churchill. Royal 8vo., pp. 493, with illustrations. Price: 15s. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE TEACHING OF PHYSICAL THERAPY.

WHAT is known as physical therapy has always been an integral part of medicine. When a medical practitioner of the past covered his patient's chest with a poultice, when he ordered hot baths for aches and pains, or cold baths to reduce fever, he was using physical therapy. In those days elaborate electrical apparatus had not been made, nor had many specialists arisen. But with the years the simple has become complex and medicine has perforce taken up the complex. During recent years physical therapy has received such adulation that it has become a specialty; and, though in certain quarters this so-called specialty is dignified, in others it has become flamboyant. Books on physical therapy abound and, according to many authors, it will cure almost anything; the claims made are often quite preposterous and, were they not made in the name of science, would be laughable. The difficulty comes when we try to discover from books what is the effectiveness of a physical agent in a particular morbid condition. The Council of Physical Therapy of the American Medical Association is concerned that a proper perspective in regard to physical agents should be maintained, and in its wisdom has authorized the publication of an article by I. S. and J. S. Cutter on the teaching of physical therapy to undergraduate medical students.[1]

From this discussion it will be well to exclude radiation therapy by X rays, radium, ultra-violet rays and so forth. These are weapons for highly trained persons who understand the physical principles underlying their use, who realize their limitations, and who know how to avoid the dangers connected with them. It is with simpler methods that I. S. and J. S. Cutter are chiefly concerned. They state that physical therapy is probably one of the most misunderstood subjects in medicine, and they think that certain medical practitioners mistrust physical therapy procedures in the treatment of patients because of their inability to advise the correct treatment. They deprecate the flooding of the market with physical therapy equipment and they state, we think quite rightly, that the practitioner is too often driven to the purchase of equipment that he knows little about. The remedy, in their opinion, is that students should know that it is not necessary to possess elaborate apparatus in order to apply physical therapy—"the best physical agents to use are usually the cheapest, the simplest and the easiest obtained". Heat, massage, exercise and water, they hold, can be used by any medical practitioner in any place if he has had proper instruction in their application. In endorsing this statement we should make it quite clear that the application of heat by diathermy is not to be looked on as outside the province of a general practitioner. At the same time it must be realized that if a practitioner is well versed in the use of simple methods, he will not have to rely so often on an elaborate machine.

In the teaching of the undergraduate student physical therapy must be approached as a part of general medicine and not as though it were a specialty; preferably it should form part of the tuition in therapeutics. Our American authors endorse the practice of giving special lectures in the subject. This is surely a mistake. To give special lectures in physical therapy will give the student the impression that he is dealing with a special subject. In his early years he will have

[1] *The Journal of the American Medical Association*, June 2, 1934.

learned all that is necessary to him as an undergraduate regarding the physical basis of even so complicated a subject as radiation therapy. If he is taught that physical agents are used in the treatment of disease in conjunction with other forms of therapy, and if he receives this instruction as part of his teaching in clinical medicine, he is more likely to have a proper perspective. The practical application of various agents can be studied in an out-patient department, and this department should be part of the medical unit of the teaching hospital. The application of certain forms of physical therapy in general practice will be discussed on another occasion. For the present we must be content to argue that if the undergraduate student is taught the correct relationship of physical therapy to other forms of treatment, he will not be induced to buy apparatus that is unnecessary, and he will not believe the extravagant claims made by the authors of the flamboyant school.

Current Comment.

THE ADRENOGENITAL SYNDROME.

VIRILISM in women associated with cortical adrenal tumours is well known. Such patients may have hirsuties, a male voice and amenorrhœa. A case is recorded of a male child of four years having hirsuties and precocious sexual development with an adrenal cortical malignant growth and metastases in the lungs. There have been two female children aged two years reported with virilism, sexual precocity and cortical tumours. From one the growth was successfully removed, with regression of the sexual precocity.

L. R. Broster[1] reviews the subject and details the anatomy and embryology of the adrenal. In man these glands reach their maximum development at birth; the cortex then undergoes absorption and regeneration. Cortical cells are derived from the genital ridge, which gives rise also to the testis and ovary. Determination of sex takes place about the sixth week and a fuchsinophile reaction appears two to four weeks later. There is a marked connexion between this reaction and a state of maleness in the female. The substance so staining may be identical with or closely related to the male hormone. Broster suggests that a strong and prolonged male phase in the female may be a factor in determining sex. The occurrence of secondary male characters in the female has long been known. In 1905 W. Bulloch and J. H. Sequeira demonstrated the relation between the precocious appearance of secondary sex characters and cortical adrenal tumours. In this syndrome retrogression of primary and secondary feminine characters and their functions is associated with the appearance of secondary male characters. The distribution of hair accords with the male pattern. Alterations of bodily contour approximate to the male type. The female genitalia are immature and the psychological outlook has a masculine tendency. There are three main groups: (i) adrenal hermaphroditism, with changes declared at puberty; (ii) adrenal virilism, the changes appearing after normal puberty; (iii) mild virilism, the "fat type", with other endocrine disorders occurring after puberty. Broster would add a fourth group, termed post-menopausal virilism. In the first a masculizing factor is at work and, if it begin some time before puberty, it will alter the feminine figure to that of a muscular man. In the second there are other signs of endocrine imbalance. Some sufferers become fat, but in the majority this fat is lost; some drift into multiglandular types. The third is possibly a multiglandular syndrome or is perhaps due to a basophile adenoma of the pituitary associated with adrenal disorder.

Broster's investigations revealed slight changes in blood cholesterol and sugar metabolism. There was a tendency to show resistance to insulin. In the third group there was an increase in the red cell count. Histological examination elicited no abnormality in the arrangement or shape of the cortical cells. Perhaps their function was altered. H. W. C. Vines, using the Ponceau fuchsin stain, showed that in every case of unilateral adrenalectomy the cytoplasm of the cortical cells contained a vivid red granular matter, chiefly in the inner and middle zones. The colour varied in intensity, suggesting that the response was both qualitative and quantitative in the cases of hyperplasia. Portions removed from normal adrenals during kidney operations, and normal adrenals taken at autopsies all failed to take the stain. Adrenal tumours associated with virilism were positive. The fuchsinophile material in the cells survives *post mortem* conditions. Vines obtained a strong reaction in the male fœtus between the ninth and seventeenth week. In the female fœtus it was weaker and more brief, lasting from the eleventh to the fourteenth week. After the twentieth week it was absent in both sexes. In the embryo of both sexes the reaction was early and evanescent. In other tissues traces of the reaction were seen in the interstitial cells of the testis, the *corpus luteum* and the anterior lobe of the pituitary. A positive reaction has been noted in true hermaphroditism. The cells of the adrenal cortex have the power of developing this substance in adult life, and it may become activated at periods of physiological stress, as at puberty. The first and second groups may be thus accounted for, but the third group is not so simply explained. Broster suggests that in adult female virilism there is a failure on the part of a feminizing influence to counteract the male phase. The brief duration of such male phase in the female fœtus suggests that it is suppressed, such suppression being due to a

[1] *The Lancet*, April 21, 1934.

mechanism depending on other endocrine activity. Vines found in two female fœtuses a prolonged male phase accompanied by pituitary hyperplasia. Virilism may be due to failure to maintain a normal balance between the anterior pituitary lobe and the male adrenal. Pituitary dysfunction would occur and the clinical picture would depend on the predominance of either factor. With pituitary dysfunction in the ascendant there is a shift towards the type of basal adenoma of the pituitary described by H. Cushing.

Broster, from his analysis of sixty cases, concludes that heredity plays some part, particularly on the female side. Regarding other endocrine disturbances, patients appeared whose mothers had Graves's disease or parenchymatous goitre or diabetes. Women with virilism are comparatively infertile. When conception occurs, miscarriage is common. A patient with amenorrhœa had unilateral adrenalectomy and subsequently bore a son. Broster suggests that the adrenogenital syndrome is not a disease, but a deviation from the normal evolutionary process. He states that we must think of a transient normal fœtal phase persisting and so giving rise to the abnormal in the adult. He believes it to form a definite streak in the human species, some of its minor stigmata being not uncommon. A similar syndrome occurs in the adult hen. In some of the lower animals such sex changes are common. Broster considers that, if his suggestion of the male hormone be correct, it corroborates the theory of bisexualism, at all events as far as the female is concerned. But this assumption is certainly not proved. The altered sex outlook of these patients, which becomes normal after unilateral adrenalectomy, suggests an endocrine basis for some forms of mental deviation. The problem is of great interest to the eugenist and is important in medico-legal matters. In man the presence of the syndrome is a retrograde movement tending towards the intersex type. Broster asks whether it is stationary, receding or increasing. If it is increasing, the evolutionary process is drifting to a neuter state. He concludes with interesting but contentious speculations concerning the bearing of the condition on our economic and social problems—the emancipation of women, over-population and race deterioration.

PULMONARY LOBECTOMY.

In spite of the advances that have been made in thoracic surgery, and these advances have been considerable, pulmonary lobectomy is one of the most serious operations that can be undertaken. In an important contribution on bronchiectasis in 1932 Ballon, Singer and Graham pointed out that it was hardly fair to draw general conclusions from a list of operations performed by different operators who used different methods. They showed, however, that in 212 collected cases no less than 72 patients, or 34%, had died apparently because of the operation, and that in only 99 patients was a satisfactory result obtained. They stated that if only the recent and more favourable figures were taken, a patient with bronchiectasis who submitted to a lobectomy ran about a 15% to 20% chance of dying because of the operation, and that if he recovered from the operation he had only about a 65% chance of having a satisfactory result with solid healing of the wound. In contemplating figures such as these, it is necessary to remember that the patients who died as a result of operation were probably so ill that they would have died before long without operation, and that those who recovered were relieved of what must have been an intolerable condition and were given a new lease of life. In other words, even with such a chance of death, the risk was worth taking. Removal of the lobe of a lung is undertaken, of course, for conditions other than bronchiectasis. There is no doubt that with the greater degree of accuracy in diagnosis attainable by bronchoscopy, pulmonary lobectomy will be attempted oftener in the future.

One of the factors that have a profound bearing on the feasibility and on the success of lobectomy is the adhesion of the visceral to the parietal pleura. Particularly in bronchiectasis and in abscess of the lung the presence of adhesions is often important in preventing the spread of infection. Operation is often undertaken in several stages, and pleural adhesions are deliberately produced. It is known, moreover, that death sometimes results from the sudden and wide opening of one side of the pleural cavity. Z. Takàcs has recently discussed this question.[1] He holds that death in these circumstances is due to an alteration in the carbon dioxide balance. He opened the chests of dogs widely after he had fixed half the lung against the thoracic wall by sutures. Breathing went on unhindered. Twenty to thirty minutes later the sutures were removed and the lung was allowed to collapse; the breathing, apart from being a little deeper, was not affected. This is significant when we recall a successful removal of a lung by Graham and Singer, referred to in these pages on December 23, 1933. In this instance the lung was thrown out of action by pneumothorax for some time before it was removed.

These investigations and reports show that progress is being made in pulmonary surgery. The latest contribution comes from A. Graham Bryce, of Manchester.[2] He has produced dense adhesions in the pleural sac in animals to imitate conditions found in practice in man. He has caused gangrene of a lobe by ligating its pedicle and in some instances has successfully removed the gangrenous tissue. He holds that his findings support the proposal that it would be possible to remove densely adherent lung from man by cutting off its blood supply and allowing it to slough, the extraction of the dead lung being deferred till it had separated by the formation of a line of demarcation between it and its surroundings. If this could be done in man, it would allow lobectomy to be undertaken when otherwise it would be quite impossible.

[1] *Der Chirurg*, January 1, 1934, abstracted in *The Australian and New Zealand Journal of Surgery*, July, 1934.
[2] *The British Journal of Surgery*, April, 1934.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Pulmonary Fibrosis of Hæmatite Miners.

M. J. STEWART AND J. S. FAULDS (*The Journal of Pathology and Bacteriology*, July, 1934) have examined the lungs of fifteen miners who died as a result of a grossly fibrotic lesion of the lungs, in which both silica and hæmatite dust are present in large amount. The lesion is characteristic mainly on account of the hæmatite dust present; the densely fibrosed areas assume a bright brick red colour. The fibrosis is diffuse rather than nodular; it is often massive and always most intense in the upper half of each lung. An associated or superimposed tuberculosis was present in eleven of the fifteen cases. The silica content of the lungs in fourteen cases averaged 1·66% of the dry weight. This is compared with 1·72% for a group of eight cases of ordinary silicosis and 1·78% in a series of sand-blasters examined by the same method. The authors discuss the incidence of the condition in relation to the type of drilling used in the mines and the frequency of blasting.

Neurogenic Tumours of the Sympathetic System.

JOHN W. S. BLACKLOCK (*The Journal of Pathology and Bacteriology*, July, 1934) discusses neurogenic tumours of the sympathetic system in children. He found that of a series of malignant tumours in children those arising from the sympathetic system were fourth in order of frequency. In the eighteen tumours observed by him different stages of development of the cells were observed from one composed of the most primitive type of sympathetic cells (sympathogonia) through an intermediate group in which many of the sympathogonia had differentiated into sympathoblasts and small ganglion cells, to one composed only of mature ganglion cells. In certain tumours transitional stages could be traced from small embryonic cells to small and mature ganglion cells. He has suggested the following scheme for the classification of growths according to the differentiation of their cells. Group I comprises sympathicoblastomata (all malignant). This is subdivided into (*A*) undifferentiated and (*B*) differentiated groups. The undifferentiated tumours are composed only of sympathogonia. The differentiated tumours comprise: (i) those composed of sympathogonia and sympathoblasts, (ii) those composed of ganglion cells in addition to more primitive cells (ganglio-sympathicoblastomata). Group II comprises ganglioneuromata (generally simple) and these are composed of mature ganglion cells only. A possible third group might comprise tumours arising from phæochromoblasts and phæochromocytes. The tumours observed by the author all occurred in children between the ages of four months and seven years. He did not find that the age factor influenced the degree of differentiation. The author refers to Landau's suggestion that the malignancy of these tumours tends to decrease *pari passu* with advancement in differentiation of the tumours cells and increasing age of the host. He gives several reasons for regarding this view as incorrect. He also holds that there is no justification for the division of sympathico-blastomata into Hutchison and Pepper types adopted by some authors according as skull metastasis or enlargement of the liver is present.

Immunity to Induced Tumours.

THE injection of mouse blood into a mouse induces resistance against the growth of a tumour introduced by inoculation. The injection of mouse embryo emulsion has the same effect. Embryo skin is the most effective tissue in inducing resistance to tumour inoculations. The injection of embryo skin causes no immunity against the development of spontaneous tumour. P. C. Pybus (*The British Journal of Experimental Pathology*, April, 1934) has carried out experiments to discover whether it was possible by injections of embryo skin to produce an immunity to the development of tumours induced by tar painting. He describes in detail the methods used. He found that the development of tar tumours was neither prevented nor delayed after various numbers of injections. Injections of mouse embryo skin, continued after the appearance of tar tumours, had no curative effect. The numbers of metastases found in the control mice were so much greater than those found in the inoculated mice that it is suggested that the inoculations of skin, although useless for producing an immunity to induced tumours, do develop a resistance in the animal against the metastatic spread of malignant cells.

Hodgkin's Disease.

PAUL E. STEINER (*Archives of Pathology*, June, 1934) has investigated diseased splenic and lymph glandular tissue from fifteen patients with Hodgkin's disease, together with control diseased tissues from eight patients with other lymphomata. He refers to the work of Uts and Keatinge, published in THE MEDICAL JOURNAL OF AUSTRALIA. The author investigated: (i) the ability of the diseased tissue to produce tuberculosis in chickens, guinea-pigs, rabbits, dogs and mice; (ii) the disease-producing capacity of these tissues as grafts; (iii) the presence of tubercle bacilli or atypical acid-fast bacteria in these tissues. The diseased tissues were transplanted into 199 animals. The criteria used for the diagnosis of tuberculosis in the animals following injection were: (i) the acquisition of a positive reaction to tuberculin, (ii) the gross and microscopic morphological structure of tuberculous lesions, (iii) the presence of stainable acid-fast bacilli in suspicious lesions, (iv) the growth of acid-fast bacilli from such lesions on culture media, (v) the production of tuberculosis in animals in passage experiments. Tuberculosis occurred in one chicken following injection of material from a patient with Hodgkin's disease, and in one guinea-pig following injections of lymphosarcomatous tissue. In the infected chicken a spontaneous infection was not excluded. In passage experiments from these infected animals tuberculosis was again produced. Numerous lesions in diseased chickens superficially suggesting tuberculosis were regarded as non-tuberculous because they did not definitely satisfy any of the five criteria laid down. No evidence was found that the diseased human tissues were transplantable or that they were capable of inducing in animals lesions with a similar histological structure. No strains of acid-fast bacilli were grown from these diseased human tissues by cultural methods. Likewise the occurrence of acid-fast forms of bacteria reported to exist as a transient phenomenon early in the cultures of these tissues was not confirmed. Avian tubercle bacilli detectable by the methods used were apparently not present in the tissues of the fifteen patients with Hodgkin's disease.

Ischæmia and the Development of Tumours.

JOHN W. ORR (*The British Journal of Experimental Pathology*, April, 1934) has produced scar tissue in the subcutaneous tissues of mice by the insertion of linen thread sutures; the sutures were subsequently removed and healing was allowed to take place. Care was taken to avoid direct injury to the epithelium. Tar applications induced tumours more rapidly in these mice than in controls: the percentage of carcinoma at the twenty-first week was twice that in the controls. Local injection of vaso-constrictor drugs produced an acceleration in tumour induction; this was more pronounced with adrenaline hydrochloride than with ephedrine sulphate. Tumours appeared more rapidly than usual when tar was applied after ephedrine sulphate injections were discontinued. The author concludes that carcinogenic agents act upon cells which have been deprived of a fully adequate vascular supply.

Tumours of the Abdominal Wall.

L. HORN (*Monatsschrift für Geburtshülfe und Gynäkologie*, May, 1934) discusses the origin of swellings of the abdominal wall which are apparently of inflammatory origin and which always occur in the region of the scar of some operation, especially that of ventro-suspension of the uterus. These were first clearly described by Schloffer, whose histological findings were: fibrous connective tissue with small cell infiltration, eosinophilia and the formation of new blood vessels, but no evidence of malignant disease. In many of such cases the

induration and fistula formation resemble an actinomycotic infection, but the characteristic organisms are not found.

Syphilitic and Rheumatic Disease of the Aortic Valve.

R. V. SAGER AND A. R. SOHVAL (*Archives of Pathology*, June, 1934) report three cases of combined syphilitic and rheumatic disease of the aortic valve. As far as they are aware, this association has not been previously reported. They lay emphasis on the limitation of syphilitic changes in the aortic leaflets to their commissural extremities in the great majority of cases and on the diffuse nature of the characteristic lesions of rheumatic valvulitis.

MORPHOLOGY.

Musculus Thyreo-Trachealis.

NIKOLAUS TURKEWITSCH (*Folia Anatomica Japonica*, June, 1934) gives an account of an anomalous muscle attached above to the inferior border of the thyreoid cartilage and near the mid-line, and below, to a linear attachment on the opposite side, extending from the upper border of the first cartilaginous ring of the trachea to the lower border of the second. It lies immediately superficial to the *cricothyreoideus*. The author calls it the *musculus thyreo-trachealis*.

The Development of Blood and Blood Vessels in the Chorion of Man.

B. H. JÄGERROOS (*Acta Societatis Medicorum Fennicae "Duodecim"*, Series B, Volume XIX, 1934) describes investigation undertaken to ascertain whether early vasculogenesis, including blood formation, exists in the chorion of man. The main results of the investigation are as follow: Early forerunner stages, occurring in abundance, lead in unbroken sequence from the undifferentiated chorionic mesenchyme to the already known later forerunners of the primitive red blood corpuscles. The author demonstrates two plainly distinguishable cell strains of the primitive red blood corpuscle which succeed one another. These are called ichthyoidic and sauroidic cells owing to their more or less resemblance to the erythrocytes of the ichthyopsides and sauropsides respectively. When the forerunners of the ichthyoidic or sauroidic cells arrange themselves into a row, either within the fibrils of the ground stroma or independently of them, a wall unfailingly forms around the cell piles thus formed. The blood vessels form through the amalgamation of these vessel *Anlagen* into more extensive systems. Provided the young cells do not arrange themselves as mentioned above, they develop extravascularly. The author concludes that the primitive red blood corpuscles in a very early stage (during the latter half of the third week) chiefly originate from the mesodermal nuclei of the chorionic stroma. Their development always shows the same forerunner stages, which, until the middle of the second embryonal month, almost always end as ichthyoidic cells. The sauroidic cells, which occurred before that time in slowly increasing numbers, thereupon take the place of the ichthyoidic cells now rapidly disappearing. By the end of the second month secondary erythrocytes appear among the sauroidic cells, which just between the third and fourth months in their turn completely disappear, leaving the secondary erythrocytes a free field of action. The blood vessels arise from vessel *Anlagen* which develop as above described in connexion with the forerunners of the red blood corpuscles. The vasculogenetic potentialities, which are almost ubiquitous in quite young eggs, are even more inactivated in older eggs, whereby there come to stand against each other two plainly distinguishable zones, an active and an inactive zone. Finally, by the beginning of the fourth month *in utero* the activity in the entire chorionic region is paralysed and transferred into other hæmopoietic organs, which have already previously taken over this activity.

The Innervation of Striated Muscle.

H. J. WILKINSON (*Journal of Comparative Neurology*, April, 1934) gives an account of experiments which consisting of a repetition of those done by Boeke and others which have been put forward as evidence of the sympathetic innervation of striated muscle. The sympathetic nerves were isolated by section of the somatic nerves and their subsequent degeneration in the intercostal and interosseous and lumbrical muscles of cats. The sympathetic nerves were found only in relation to the blood vessels and no evidence was found in support of the hypothesis that striated muscle fibres are innervated by both somatic and sympathetic nerves. Further, no evidence was found in favour of the existence of a neurofibrillar peri-terminal network which could be regarded as a transition between the neurofibrils of the motor axone and the substance of the muscle fibre. The author also discusses "Remarks" recently published by Boeke in the same journal.

The Number of Glomeruli in the Human Kidney.

T. KOHDA (*Japanese Journal of Medical Sciences*, I, *Anatomy*, Volume IV, June, 1934) states that the number of glomeruli in the human kidney (in 21 Japanese, 30 kidneys), in ages ranging from four days to seventy-nine years, amounts to from 394,079 to 1,082,494. He further determined that the number of glomeruli is somewhat less in the Japanese than in the European and American, and that the number in old age is one-third to one-half of that in the prime. Also the number in the new-born is not very different from that in the adult, and the number in the right kidney is about the same as that in the left. The number, too, does not vary much according to sex.

The Number of Glomeruli in the Kidneys of Animals.

T. KOHDA (*Japanese Journal of Medical Sciences*, I, *Anatomy*, Volume IV, June, 1934) gives the result of counts made in various species, including the ox, horse, pig, cat, dog, rabbit, guinea-pig and monkey.

Ratio of Vascular Space to Bone Tissue in the Compact Substance of the Human Long Bone.

K. OHKUBO (*Japanese Journal of Medical Sciences*, I, *Anatomy*, Volume IV, June, 1934) finds that the percentage of the vascular area to proper bone tissue in human long bones varies from 4% to over 20%, the highest average being about 11%. The bone of the young contains more blood vessels than that of the old, and the vascular area is largest in the humerus and the fibula. The vascularity varies in different parts of the bone, being higher in the upper parts. Also there are more vessels which enter from the inside than from the outside. On comparing bone tissue, connective tissue and cartilage, the author concludes that bone tissue has more blood vessels than the other tissues.

Nuclear Configuration of the Thalamus of Macacus Rhesus.

R. L. CROUCH (*Journal of Comparative Neurology*, June, 1934) states that the most striking thing about a comparative study of the thalamus is the difference in relative size of the groups of nuclei among animals in different levels of the mammalian scale. There is a difference in the lateral nuclei, even in the lower and the higher primates. This difference is greatly augmented in the span between the rodents and the primates. On the other hand, there are other groups, the medial and the anterior, which attain their maximum development in lower forms and show relatively no increase as the mammalian scale is ascended. The author gives an account of the various nuclear masses as seen in the *Macacus rhesus* monkey.

The Vertebrate Nervous System.

A. N. BURKITT (*Proceedings of the Linnean Society of New South Wales*, May, 1934) gives an account of the progress which has been made in recent years in gaining a more rational conception of the nervous system. He shows how this has been greatly aided by researches upon the nervous system of the lower vertebrates. Among the topics he discusses are the influence of vision upon the evolution of the nervous system, the lower reflex mechanisms, the thalamus and the cerebral cortex.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Theatre, University of Adelaide, on June 27, 1934, DR. E. BRITTEN JONES, the President, in the chair.

Annual Report of Council.

The annual report of the Council was received and adopted. The report is as follows.

Election.

At the annual meeting held last June the following were elected:

President: E. Britten Jones.

Vice-President: Leonard C. E. Lindon.

Honorary Medical Secretary: Alan H. Lendon.

Honorary Treasurer: F. St. John Poole.

Members of Council: G. H. Burnell, A. R. Clayton, and D. R. W. Cowan.

Members of Federal Council: Sir Henry Newland and Bronte Smeaton.

At the first meeting of the Council held on July 6, 1933, the following subcommittees were appointed:

Scientific: The President, G. H. Burnell, A. R. Clayton, D. R. W. Cowan, L. C. E. Lindon (Honorary Secretary, Convener).

Contract Practice: The President, H. Halloran, F. St. John Poole, A. F. Stokes (Lay Secretary, Convener).

Library: The President, G. H. Burnell, Allan D. Lamphee, Sir Henry Newland (Honorary Secretary, Convener).

Parliamentary Bills: The President, L. C. E. Lindon, F. St. John Poole (Lay Secretary, Convener).

Revision of Rules: The President, Sir Henry Newland, Bronte Smeaton (Lay Secretary, Convener).

Ethical: The President, L. C. E. Lindon, Sir Henry Newland, F. St. John Poole, Bronte Smeaton, A. F. Stokes (Honorary Medical Secretary, Convener).

Meetings.

The Council.—The Council met on fifteen occasions, the attendance being:

G. H. Burnell	11	L. C. E. Lindon	11
A. R. Clayton	11	Alan H. Lendon ...	14
D. R. W. Cowan ...	14	Sir Henry Newland .	12
H. Halloran	15	F. St. John Poole ..	13
E. Britten Jones ..	15	Bronte Smeaton	15
Allan D. Lamphee ..	10	A. F. Stokes	13

Scientific Subcommittee.—The Scientific Subcommittee met once, the attendance being:

G. H. Burnell.	E. Britten Jones.
A. R. Clayton.	L. C. E. Lindon.
D. R. W. Cowan.	Alan H. Lendon.

Contract Practice Subcommittee.—The Contract Practice Subcommittee met on eleven occasions, the attendance being:

H. Halloran	11	F. St. John Poole ...	11
E. Britten Jones ...	10	A. F. Stokes	11

Library Subcommittee.—The Library Subcommittee met on two occasions, the attendance being:

G. H. Burnell	2	Allan D. Lamphee ...	1
E. Britten Jones ...	2	Sir Henry Newland ..	1
Alan H. Lendon	2		

Revision of Rules Subcommittee.—The Revision of Rules Subcommittee met on two occasions, the attendance being:

E. Britten Jones	2	Bronte Smeaton	2
Sir Henry Newland ..	2		

Parliamentary Bills Subcommittee.—The Parliamentary Bills Subcommittee met on three occasions, the attendance being:

E. Britten Jones	3	F. St. John Poole ...	3
L. C. E. Lindon	3		

Ethical Subcommittee.—The Ethical Subcommittee met once, the attendance being:

E. Britten Jones.	F. St. John Poole.
Alan H. Lendon.	Bronte Smeaton.
L. C. E. Lindon.	A. F. Stokes.

Monthly General.—Nine meetings were held, the interest and attendance being well maintained. The September meeting was held at Moonta, thirty-five members being present. The Council appreciates the assistance given by A. R. Clayton in connexion with this meeting.

The following programme was carried out during the year:

1933—

July.—Paper by C. T. de Crespigny: "Some Observations on Some of the Rarer Forms of Anæmia." (Discussion opened by E. McLaughlin.)

August.—Paper by John Mayo: "Some Aspects of Modern Radium Treatment." (Discussion opened by B. S. Hanson.)

September.—Paper by H. R. Pomroy: "The Treatment of Appendicitis." (Discussion opened by Ivan Jose.)

October.—Paper by R. M. Glynn: "Œsophageal Obstruction." (Discussion opened by H. A. McCoy.)

November.—Paper by Guy Lendon: "The Significance of Systolic Murmurs." (Discussion opened by C. F. Pitcher.)

1934—

February.—Paper by Leonard C. E. Lindon: "Some Remarks on the Diagnosis of the Acute Abdomen." (Discussion opened by G. H. Burnell.)

March.—Paper by G. H. Barham Black: "Migraine." (Discussion opened by F. H. Beare.)

April.—Clinical evening at the Children's Hospital.

May.—Listerian Oration by Sir Henry Newland: "Lord Lister: Certain Aspects of his Life, Work and Character."

Listerian Oration.

The oration this year was delivered by Sir Henry Newland on May 31, his subject being "Lord Lister: Certain Aspects of his Life, Work and Character". An excellent attendance was recorded, and the Council desires to thank Sir Henry Newland.

At the close of the oration the President entertained the members at supper in the refectory.

Membership.

The membership of the Branch is 359. The number of new members elected was twenty-one, the balance representing the difference between transfers "in and out" after deducting deaths *et cetera*. It is with deep regret that we record the death of the following members: A. W. Hill, H. W. Hodgson, W. D. MacGillivray, and Sir Joseph Verco.

Representation on Boards.

Metropolitan Infectious Diseases Hospital Board.—P. T. S. Cherry has been appointed for a further term.

Medical Board of South Australia.—H. H. E. Russell has been nominated for a further term of two years.

Work of the Sections.

Eye, Ear, Nose and Throat Section.—Six meetings were held during the year, the attendances being satisfactory.

Clinical Medicine Section.—Six meetings were held, the average attendance being twenty-nine.

Anæsthetics Section.—Three meetings were held, and there has been a marked increase in membership.

History of Medicine Section.—Four general meetings were held during the year, the average attendance being nine.

Surgery Section.—Four clinical meetings were held, the average attendance being twenty-five.

Lodge Practice.

Lodge matters have necessitated close attention during the year, the Contract Practice Subcommittee having met on eleven occasions. Every effort has been made by the Council to maintain the conditions laid down in the New Model Lodge Agreement, but the continued financial stress has made the position very difficult. Many lodge members have been unable to keep up their lodge payments, and this has been reflected in the lists of lodge surgeons. Conferences have been held with the representatives of the lodges, who stated that, owing to the demands made on their funds for sick pay and medicine, the position was becoming very serious, and unless some relief was obtained in this respect it would be impossible for some of the lodges to carry on. The Council has asked the lodge surgeons to assist the lodges in this respect as far as possible. Although attempts have been made by the lodges to secure a reduction in the agreed rates of medical fees, no alteration has been made, the rate remaining the same as during the past six years.

Adelaide Permanent Post-Graduate Committee.

The annual refresher course was held from May 28 to June 2 last. The "E. C. Stirling" Lectures were delivered by S. O. Cowen, of Melbourne. A large number of members took advantage of the lectures.

Australasian Medical Congress—Fourth Session.

The Fourth Session of Congress was held at Hobart from January 15 to 20 last. A representative number of members of this Branch attended. The arrangements for the scientific meetings and the social entertainment of visitors were admirably carried out by the Tasmanian Branch.

Control of Tuberculosis in South Australia.

As a result of the activities of the Council, the erection of the new tuberculosis clinic in the Adelaide Hospital grounds is nearing completion. Following the report and recommendations made by the Subcommittee, which were forwarded to the Government, a deputation consisting of the President (E. Britten Jones), A. V. Benson (Chairman of the Subcommittee), C. T. de Crespigny (Chairman of the Honorary Staff of the Adelaide Hospital), and H. Gilbert (Chairman of the Honorary Staff of the Children's Hospital), waited on the Chief Secretary on May 22 last and urged the necessity for centralization of control of the efforts being made to check the spread of tuberculosis in South Australia. The Government was asked to give effect to the recommendations of the Subcommittee that there should be appointed an advisory committee comprising the Inspector-General of Hospitals, the Chairman of the Central Board of Health, and a medical practitioner with special experience nominated by the British Medical Association. The Government has promised to consider the request.

Revision of Rules.

For some years the Branch has felt the need of an up-to-date copy of the rules and by-laws. The Rules Subcommittee, after careful consideration, submitted a draft copy of rules and by-laws, which, after approval of the Association solicitor, were referred by the Council to a special general meeting of members. Several amendments were adopted, and the amended rules and by-laws will now be sent to England for the approval of the Parent Association. It is expected that a suitable booklet of the rules and by-laws will be prepared containing, in addition, information of value regarding ethical matters *et cetera*.

The Late Sir Joseph Verco.

The death of Sir Joseph Verco on July 29, 1933, is deeply regretted. His work in connexion with the Association is well known, and he held the position of President of the Branch in 1886-1887, and from 1915 to 1920. The Council has given earnest consideration to a fitting memorial to commemorate his work for the Association and the medical profession, and also the honour he brought to the practice of medicine in this State. Various schemes were considered, but under existing economic conditions it was felt that any memorial entailing a large expenditure, whilst desirable, was not practicable. The Council finally decided that it may be possible to set aside some portion of the contemplated new building to be erected by the British Medical Hall Company, as a tribute to his memory. When the plans for such building are available, the matter will be discussed with the Directors of the British Medical Hall Company, and at a general meeting members will have an opportunity of considering the scheme.

Hospital Policy for the Branch.

A subcommittee was appointed by the Council to deal with this question. In addition to making a report to the Federal Council, which submitted a *questionnaire* on this subject to all Branches, the Council has also considered conditions essentially local, as it is felt that the present system of administrative control might be improved.

Annual Meeting, Melbourne, 1935.

The President (E. Britten Jones) and the Honorary Medical Secretary (Alan H. Lendon) have been nominated to represent the Branch on the Executive Committee of the annual meeting of the Parent Association to be held in Melbourne during September, 1935.

Hospital and Medical Services Insurance.

The scheme of the Colonial Mutual Life Assurance Company which had been adopted in other States was approved by the Council after several conferences with representatives of the Company, and should benefit members. The scheme is intended to relieve the financial difficulties of public hospitals and at the same time guarantee to the medical profession a measure of recompense for their services. Over 1,000 policies have been taken out up to the present time.

Workmen's Compensation Act.

The Council has considered the question of additional clauses to the *Workmen's Compensation Act* to provide for payment of medical and hospital expenses to injured insured persons. The subcommittee appointed to inquire into the matter has submitted its report to the Council, and it is thought that the most tactful method of approach to attain the desired amendments to the Act would be for the subcommittee to meet informally three representatives of the industrial insurance companies in order to ascertain their views and endeavour to enlist their support. A conference is being arranged accordingly.

Medical Officers to the Destitute.

In February, 1931, the Council approached the Government in an endeavour to secure better conditions for medical officers, but owing to the financial depression the Government could not do anything in the matter. However, in view of the enormous increase in the work now being performed, it was felt that many of the medical officers were rendering this service at considerable financial loss to themselves. The Council feels that the cost of this work should be borne by the whole State and not by one section of the community. A subcommittee was appointed to again inquire into the matter, and from the information obtained the financial loss incurred by members in connexion with this work was confirmed. Strong representation has recently been made to the Government by the Council and recommendations made in order to secure adequate and equitable remuneration. The matter is now being considered by the Government.

Influx of European Medical Practitioners.

Correspondence has passed between the Council and the Federal Council regarding the probable influx of European medical practitioners into Australia. Copies of the letters were passed on to the Medical Board of South Australia, who have advised that the Board viewed the matter seriously and had addressed a letter to the General Medical Council of Great Britain on the question.

Representative on Central Council in London.

Sir Thomas Dunhill, having served six years as Group Representative (which includes the South Australian Branch) on the Central Council, was not eligible for reelection for 1934, and Professor R. J. A. Berry has been nominated in his place.

Fees for Persons Injured in Motor Accidents.

A bill to make provision for payment of remuneration to registered medical practitioners and hospitals for emergency treatment rendered to persons injured through the use of motor vehicles on roads and in places to which the public have access was recently introduced into the House of Lords. The British Government has announced that it is unwilling to give facilities for the progress of the bill through the Lower House, but is willing to consider the issues raised in connexion with the increase of road accidents. Certain proposals have been made, and it is contemplated that these proposals will contain suggestions for dealing with the medical aspects of the matter.

The Council has taken the question up with the Parent Association, and when finality has been reached in London the matter will be further considered with a view to introducing in South Australia a scheme which will provide payment for such treatment.

Medical Act of South Australia.

During the year Council has had under consideration the *Medical Act* of South Australia. It was felt that means should be taken to limit the activities of unqualified persons. Sections of the *Medical Act* operating in Western Australia, if adopted in South Australia, would relieve the position in this respect, and the attention of the Medical Board has been drawn to the necessity for action in the matter, and suggesting that Section 23 of the Western Australian Act might be incorporated in the South Australian Act.

Federal Council.

Two meetings of the Federal Council were held during the year, the first on August 28, 1933, at Sydney, and the second on January 15, 1934, at Hobart. Our representatives, Sir Henry Newland and Bronte Smeaton, attended both meetings.

General Remarks.

The report indicates the main activities of the Council during the past year.

The year has been a busy one, many matters affecting the interests of members having received consideration. More than the usual amount of attention has been given to matters concerning country members. In this connexion it is anticipated that when the new rules come into force, in addition to having voting facilities provided for them in connexion with the annual election of officers, country members will always have at least two representatives on the Council.

(*Signed*) E. BRITTEN JONES,
President.

Financial Statements.

The financial statements, as submitted by the Honorary Treasurer, Dr. F. St. John Poole, were received and adopted. The statements are published herewith.

Induction of President.

Dr. E. Britten Jones then introduced the President, Dr. L. C. E. Lindon, and wished him a successful term of office.

Dr. L. C. E. Lindon thanked the members for having elected him to the presidential chair.

Income and Expenditure for Year ended December 31, 1933.

	£ s. d.	£ s. d.		£ s. d.	£ s. d.
To Amount due British Medical Association, London, on Paid Subscriptions	406 12 0		By City Subscriptions	822 19 0	
„ Amount due British Medical Association, London, on Unpaid Subscriptions	54 16 6		„ Country Subscriptions	482 17 0	
		461 8 6	„ Accrued Subscriptions	175 5 0	
„ THE MEDICAL JOURNAL OF AUSTRALIA		426 5 0			1,481 1 0
„ Library Account		55 0 0	„ Interest		34 6 11
„ Telephone	28 11 10		„ Sundries		0 4 2
„ Exchange	3 18 7				
„ Rent	52 0 0				
„ Postages and Telegrams	29 12 2				
„ General Expenses	45 7 2				
„ Stationery and Printing	24 3 3				
		183 13 0			
„ Lister Medal		0 10 0			
„ Federal Council Expenses		18 19 0			
„ Salary		350 0 0			
„ Depreciation		10 17 6			
„ Balance		8 19 1			
		£1,515 12 1			£1,515 12 1

Library Fund Account, December 31, 1933.

	£ s. d.		£ s. d.
To Adelaide University	50 0 0	By Balance brought down from December 21, 1932	516 9 4
„ Depreciation	11 5 0	„ Interest from Savings Bank	3 16 7
„ Balance	514 10 11	„ Country Subscription	0 10 0
		„ Transfer from Income and Expenditure Account	55 0 0
	£575 15 11		£575 15 11

General Fund Account.

	£ s. d.		£ s. d.
To Telephone Account for 1932	9 19 9	By Balance brought down, December 31, 1932	2,832 2 3
„ Balance	2,831 1 7	„ Income and Expenditure Account	8 19 1
	£2,841 1 4		£2,841 1 4

Balance Sheet as at December 31, 1933.

LIABILITIES.	£ s. d.	£ s. d.	ASSETS.	£ s. d.	£ s. d.
To Subscriptions paid in advance		5 11 0	By Plant and Fittings	108 13 10	
„ Amount due British Medical Association, London, on Paid Subscriptions	1,860 11 0		*Less* Depreciation	10 17 6	
„ Amount due British Medical Association, London, on Unpaid Subscriptions ..	54 16 6				97 16 4
		1,915 7 6	„ Investments—		
„ THE MEDICAL JOURNAL OF AUSTRALIA		120 0 0	Commonwealth Loan	403 12 0	
„ Library Fund		514 10 11	British Medical Hall Company, Limited	1,950 0 0	
„ British Medical Association, London, Savings Bank Interest		86 2 8			2,353 12 0
„ University of Adelaide		50 0 0	„ Library Fund—		
„ Sundry Creditors		1 0 0	Delineascope	45 14 11	
„ National Bank of Australasia, Limited		30 2 4	Projector	64 7 0	
„ General Fund		2,831 1 7		110 1 11	
			Less Depreciation	11 5 0	
					98 16 11
			„ Savings Bank Library Fund ..		132 3 0
			„ Lister Medals and Dies		19 3 4
			„ British Medical Hall Company, Limited, Loan		450 0 0
			„ Subscriptions owing		232 3 0
			„ Stocks—		
			Hospital Forms	0 15 7	
			Medical Certificate Books	3 18 11	
			Stationery	10 0 0	
					14 14 6
			Sundry Debtors		117 12 7
			„ Cash—		
			State Savings Bank	1,030 0 0	
			Commonwealth Savings Bank	910 8 10	
			In Hand	97 5 6	
					2,037 14 4
		£5,553 16 0			£5,553 16 0

F. ST. JOHN POOLE, Honorary Treasurer.
WALTER C. DOBBIE, Lay Secretary.

Certified correct.

C. W. L. MUECKE,
Chartered Accountant (Aust.),
Auditor.

Adelaide,
May 29, 1934.

Election of Office-Bearers.

Office-bearers for the ensuing twelve months were elected as follows:

Vice-President: D. R. W. Cowan.
Honorary Treasurer: F. St. John Poole.
Honorary Medical Secretary: Alan H. Lendon.
Members of Council: R. A. Haste, A. F. Hobbs, R. D. Hornabrook, L. A. Wilson.

President's Address.

Dr. E. Britten Jones, the retiring President, then delivered his address (see page 273).

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on June 28, 1934, DR. A. J. COLLINS, the President, in the chair.

Endocrine Disorders.

DR. LORIMER DODS read a paper entitled "Adenohypophyseal Dystrophies of Infancy and Childhood" (see page 277).

PROFESSOR C. G. LAMBIE gave a lantern demonstration illustrating the clinical syndromes resulting from the presence in increased, diminished or normal amounts of the growth, gonadotropic and thyreotropic hormones of the pituitary gland. The illustrations were taken from classical papers on the various syndromes as well as from Professor Lambie's own cases. He then proceeded to explain the possible reasons for the absence of certain syndromes from the theoretical number of combinations of the above factors. The nature of the anatomical and physiological connexions between the hypothalamus and the pituitary was then illustrated and discussed.

DR. LAURENCE HUGHES said that he agreed with Dr. Dods that in many children with obesity of the pituitary type the obesity was temporary—it disappeared spontaneously without any treatment at all. He referred to one family that he had attended for fifteen years. In this family

there were five male children and one female. Four of the males in turn had begun to increase in weight on account of the deposition of fat, but they were otherwise normal. The deposition of fat was of the Fröhlich type. During the adolescent period three of these children had definitely begun to lose weight—two at sixteen years of age and one at eighteen. They all became normally proportioned. In one instance it was interesting to note that when the amount of fat began to become less, the child began to have epileptic attacks. All possible tests that might reveal the presence of an intracranial lesion had been undertaken, but without result. He had also seen a child at the Royal Prince Alfred Hospital who suffered from epileptic attacks and adiposity, but in whom no intracranial lesion or other abnormality was present. Dr. Hughes referred to the fact that epilepsy often occurred in acromegaly, but asked whether the readers of the papers had ever observed the onset of epileptiform attacks associated with the disappearance of obesity of the pituitary type. He concluded by saying that the mother of the children to whom he had first of all referred suffered from obesity. He thought that heredity was a factor in many cases.

DR. A. W. CAMPBELL said that he had not intended to speak, but that in response to the request from the President he would make a few remarks. In such a discussion one necessarily had to be selective, and in these circumstances he referred to precocious puberty. He said that the syndrome, as it was seen clinically, was clearly cut and yet the pathology seemed to be diverse. Cases were known in which tumours of the ovary had been found; in other instances tumours of the suprarenal body were present, and again in others tumours of the brain. All these diverse states seemed to produce the same clinical conditions, and he asked how this was to be explained. Dr. Campbell said that he was interested in tumours of the brain associated with precocious puberty more than in other types of tumour, and he found that these did not always affect the pituitary gland. In two cases that had come within his experience the tumours were well above the pituitary body and it was impossible to say that either the pituitary or its connexions were involved and likewise the pineal gland had escaped. In two other instances the pathological change was situated near the pituitary, in the infundibulum, but at the same time the pituitary gland appeared to be normal. Such tumours might find an embryological explanation. Rathke's pouch was a kind of no-man's land, where epiblast, mesoblast and hypoblast all met. It seemed quite possible that as a result of some developmental anomaly mesoblast was transferred to the upper regions, between the pituitary gland and the pineal body. He thought that it was not unlikely that the cells of tumours might be capable of pouring out substances that might produce such a condition as hypergonadism. This idea was feasible, because it was known that the suprarenal body, the ovary and the testis were developed from mesoblasts. He asked the readers of the papers whether they had any ideas which would explain the diversity of the pathological findings.

DR. F. A. E. LAWES said that he had enjoyed both papers very much. He thought that minor degrees of pituitary dysfunction were probably very common. He was interested in what Dr. Dods had said about the disappearance of obesity. He called to mind a woman who had been shown two years previously on account of her obesity at a meeting of the local medical association at Hornsby. In the two years that had elapsed since the meeting she had manifested considerable improvement—she had lost a great deal of her obesity and her mentality had become brighter. In regard to the remarks of Dr. Hughes about acromegaly and epileptiform seizures, he called to mind a man so affected in whom X ray examination, examination of the blood and the application of the Wassermann test had all failed to reveal any abnormality; the man, however, had definite impairment of the renal function.

DR. R. A. NOBLE thanked both speakers for their papers and referred to the transient nature of many of the syndromes. They were accustomed to look on these syndromes as the permanent entities described in the text books. Some of them, however, did disappear, and in these circumstances there was often a definite relationship to epileptiform seizures. He had known patients with pituitary dystrophy who developed epileptiform seizures at puberty; the seizures subsequently disappeared. In these cases, although a search was made for a neurological condition, no evidence of one could be found. Dr. Noble thought that these manifestations were due to transient pathological changes in the pituitary gland.

DR. E. H. STOKES said that there was but little left to discuss. He agreed with Dr. Dods that few of the extracts used in the treatment of pituitary dystrophy were potent. He thought that when potent extracts were available they would be able to differentiate clinically between the different syndromes. He would like to hear more of what the speakers thought of the value of placental extracts.

DR. OLIVER LATHAM supported Dr. Campbell's remarks regarding symptoms arising in association with tumours that apparently left the pituitary region untouched. Gordon Holmes had reported three such cases. These were tumours of the hypothalamic region. Dr. Latham had had the good fortune to see a similar case with Dr. Nowland and the late Professor Hunter. The patient was an adult who developed a Fröhlich's syndrome due to a tumour of the hypothalamic region and a normal pituitary.

Dr. Dods, in reply, suggested that possibly the epileptiform seizures were due to a neuro-hypophyseal disturbance.

Professor Lambie, in reply, said that they knew very little of the mechanism by which one gland stimulated or depressed another. He referred to the difficulty of reproducing the syndromes in the laboratory and exemplified his remarks by referring to attempts to reproduce exophthalmic goitre by the use of thyroxin. With thyroxin exophthalmos could not be produced. On the other hand, it had been produced by the injection of pituitary substance. Perhaps progress would be possible when something was known of the chemical constitution of the hormones. Referring to the contention that tumours of the hypothalamic region might produce effects without changes in the pituitary gland being detected, he said that it would be impossible to determine whether a pituitary gland was normal, because there were no means of determining the changes in its circulation.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Liebert, Amanda Annie Gertrude, M.B., B.S., 1924 (Univ. Melbourne), 131, Mitcham Road, Mitcham.
McKillop, William John, M.B., B.S., 1930 (Univ. Melbourne), 283, Moreland Road, Coburg, N.13.
McLennan, Simon, M.B., 1914, Ch.M., 1928 (Univ. Sydney), Repatriation General Hospital, Caulfield.
Niall, Desmond Paul, M.B., B.S., 1932 (Univ. Melbourne), 20, Hampton Street, Hampton.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association announces that the following books have been added to the library of the New South Wales Branch: "The Races of Man", R. B. Bean; "How You Began", A. Williams-Ellis; "The Hygiene of Marriage", I. E. Hutton; "Light Therapy", F. H. Krusen; "Diabetes: Reasons and Recipes", E. E. Claxton and L. Burdekin; "Vaccine Therapy in Acute and Chronic Respiratory Infections", H. T. Gillett; "Bright's Disease", J. N. Cruickshank; "Clinical Examination of the Nervous System", G. H. Monrad-Krohn; "Modern Inhalation Therapy", C. H. Auty; "The Cure of Hæmorrhoids, Varicose Veins, etc.", S. McAusland; "The Mastery of Sex through Psychology and Religion", L. D. Weatherhead; "The Culture of the Abdomen", F. A. Hornibrook; "Clinical Contraception", G. M. Cox; "Radiotherapy in the Diseases of Women", M. Donaldson; "Recent Progress in Medicine

and Surgery, 1919-1933", Sir John Collie; "Modern Aspects of Gastro-Enterology", M. A. Arafa; "A System of Clinical Medicine", T. D. Savill; "1933 Year Book of Radiology", C. A. Waters and I. I. Kaplan; "History of the University of Edinburgh", A. L. Turner; "Electrosurgery", H. A. Kelly and G. E. Ward.

CRICKET IN MELBOURNE.

THE Social Committee of the Council of the Victorian Branch of the British Medical Association is arranging a series of cricket matches with the public schools, the university colleges and the chemists. It is proposed that these matches shall be played on successive Wednesdays throughout the ensuing season.

Those willing to take part are asked to communicate with the Secretary of the Branch, who will then summon a meeting of players and discuss details.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on July 11, 1934, DR. H. C. COLVILLE, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Rickets.

DR. M. L. POWELL showed a male child, aged two years and three months, whom he had first seen one year previously because of the child's inability to walk, although he could crawl quite freely. The child was otherwise apparently well. He had been a full-time baby, weighing 4·0 kilograms (nine pounds) at birth, and was entirely breast-fed for the first six months. He was then fed on Nestlé's condensed milk, one part of milk to eight parts of water; and biscuits, bread and butter, porridge, and mashed potatoes were soon added. He was given one teaspoonful of cod liver oil about three times per week till the age of one year, at which time both the Nestlé's milk and cod liver oil were stopped and he was fed on ordinary baby diet, including cow's milk.

There were eleven other children in the family, all of whom were normal. Home conditions were reasonably hygienic. Examination of the child revealed a surprising degree of florid rickets. Craniotabes, enlargement of the epiphyses, especially at the lower end of the radius, the tibia and the lower end of the femur, bowing of the lower third of the tibia, and a rickety rosary were all present in marked degree. Radiograms of the long bones showed typical rhachitic changes. For three weeks he was given 8·0 cubic centimetres (two drachms) of cod liver oil emulsion three times a day, and the mother was instructed regarding heliotherapy and general hygiene. He was then again examined by X rays and there appeared a slight improvement in the depositions of calcium in the metaphyseal region, but as yet no line of provisional calcification. He was then given one cachet three times a day of "Tricalcine" and irradiated ergosterol. The improvement was truly dramatic, and radiograms taken every twelve days showed deposition of calcium and a definite line of provisional calcification. At the end of three months the radiographic appearances were those of completely healed rickets. The child soon began to walk and was now practically normal.

Dr. Powell showed the patient because it was rare to see such a severe degree of rickets in a child of two years and three months with so little climatic, hygienic, or dietetic influences to account for it. He thought the most important factor was the diet, consisting almost solely of Nestlé's milk during the second six months of life. The other reason why he showed the case was that it demonstrated the striking superiority of the preparation containing irradiated ergosterol over the cod liver oil emulsion.

Sacro-Coccygeal Teratoma.

DR. H. DOUGLAS STEPHENS showed a child of eight weeks whom he had seen soon after birth because of a large lump situated just above the buttocks in the mid-line. It was present at birth and was not increasing in size. The bowels acted regularly and the child was otherwise a normal, healthy, breast-fed baby. Examination of the lump showed it to be a fluctuant swelling the size of a large orange. It arose from the mid-line in the sacral region and appeared to have a stalk which felt like cartilage. Dr. Stephens thought that the tumour was either a sacro-coccygeal cyst or a cyst of the neurenteric canal. At operation when the child was five weeks old the cyst was dissected out and found to be closely adherent to the rectum. The stalk which had been felt was of denser tissue and was attached to the left side of the sacro-coccygeal area. Although at operation the dissection was clean and complete and the rectum was not perforated, it was noted that the rectal wall was extremely thin, and two days later a fæcal fistula formed. Despite this the wound slowly granulated and now there was practically no fæcal discharge at all. Section of the tumour showed that dermoid features predominated, skin, epithelium and appendages being present. There was a mesoblastic element in the form of unstriped muscle, and the diagnosis was sacro-coccygeal teratoma.

Intrathoracic Stomach.

DR. E. M. TYMMS showed a child, aged one and a half years, who had been admitted to hospital at the age of five weeks with a history of vomiting since one week after birth. The baby weighed 4·2 kilograms (nine pounds six ounces), having weighed 4·0 kilograms (eight pounds thirteen ounces) at birth. The child was not constipated, and although the liver edge was palpable, no pyloric tumour could be detected. However, the condition was regarded either as congenital pyloric stenosis or a mild degree of congenital duodenal atresia. The child was treated for three weeks with atropine, but no improvement in the vomiting ensued. A barium meal showed that most of the barium was retained in the thorax and this was thought to indicate either a diaphragmatic hernia or else spasm of the cardia with dilatation of the œsophagus. Further examination, however, showed a congenitally short œsophagus and a partly thoracic stomach.

The baby was at first breast-fed, with supplementary feeds of cow's milk and water. At eight months it was weaned and at the present time it was able to eat light baby diet and thickened milk food, with much coughing and some vomiting. Its nutrition was poor and it now weighed only 6·3 kilograms (fourteen pounds). The bowels were frequently constipated, alternating with some diarrhœa, while at times the child had slight attacks of hæmatemesis and melæna. Dr. Tymms showed the case as a very rare congenital abnormality.

Intracerebral Hæmorrhage.

DR. A. P. DERHAM reported the history of a boy of eleven years who had suffered from headaches of increasing severity for the previous twelve months. Ten hours before admission to hospital the child collided head on with his brother, after which he had a convulsion and became unconscious for half an hour. On admission to hospital he complained of severe headache and pain in the back of the neck. There was neck stiffness and a slight Kernig's sign, and his temperature was 38° C. (100·4° F.). Otherwise physical examination revealed no abnormality at all. The child was treated conservatively and improved. Three days later his symptoms and signs recurred more intensely than at first. A lumbar puncture revealed the fluid to be under increased pressure and to contain some altered blood. The chlorides were normal. Again he improved, but three days later relapsed as before, and a second lumbar puncture again yielded uniformly blood-stained cerebro-spinal fluid, the blood being of the altered type. He slowly improved once more, his mental state became normal, his neck stiffness and Kernig's sign disappeared and his fundi were normal. A radiogram of the skull showed no abnormality and his blood did not react to the Wassermann test.

Dr. Derham at first thought the boy had a subarachnoid hæmorrhage, but at the time of the meeting he thought the condition was a leaking aneurysm. Subsequently this boy progressed very well for two weeks, when again all his symptoms returned and he died in a short time. *Post mortem* examination revealed a massive hæmorrhage into the left lateral ventricle, which was much distended, and the foramen of Monro was dilated sufficiently to admit the index finger. There was a well marked pressure cone on the cerebellum. Dr. Reginald Webster, who performed the *post mortem* examination, thought that this implied a unilateral hydrocephalus antecedent to the hæmorrhage. The twelve months' history of headaches supported this, although he could not find a cause. There was no evidence of an aneurysm. Presumably the slight head injury was sufficient to precipitate a hæmorrhage into the already distended ventricle.

Absence of Kidney.

DR. J. W. GRIEVE showed a boy of thirteen years who had been perfectly well until three days before being seen, when he complained of feeling unwell and of abdominal pain. The pain was situated to the right of the umbilicus and in the right loin. At the onset he had a slight rigor and since then has had some frequency of micturition and slight dysuria. The night before being seen he became worse and vomited for the first time.

On examination his temperature was 37·5° C. (99·6° F.), his pulse rate 88, and there was definite resistance on the right side of the abdomen, with tenderness to the right of the umbilicus and in the right loin. The urine contained albumin and microscopically a few red blood corpuscles and numerous polymorphonuclear cells. No casts were seen. A culture of the urine grew *Staphylococcus albus* predominantly with a few streptococci and Gram-negative bacilli. The following day a definite localized mass was palpable in the right loin. He was treated with rest, free fluids, warmth to the side, and was given urinary antiseptics such as neotropine and methylene blue. His temperature slowly subsided, the urine cleared considerably, although there were still some pus cells and albumin present. When he had recovered sufficiently to be examined by X rays, he was given "Uroselectan" intravenously, and serial photographs were taken. There was no shadow of any kind on the left side, and the right pelvis appeared to be that of an enlarged disk-shaped kidney with two ureters which appeared to fuse just above the bladder. Dr. Grieve said that he presumed the left kidney to be absent. The result of the urea concentration test was normal, but as this could be so with exceedingly little renal tissue intact, he still thought the prognosis was very grave because of the abnormality of the other kidney. He was surprised that the boy had lived so long apparently symptomless.

Correspondence.

AN AUSTRALIAN ORGANIZATION AGAINST CANCER.

SIR: While realizing how much the generous amount of space which you have devoted to the Cancer Conference will help the effort to control the disease, it is, I think, unfortunate that in your editorial you should have suggested that the proposed local conferences in the several States must be regarded as "parochial". This as a matter of fact is just what they are not intended to be. The Queensland Cancer Trust, which, as its Secretary, I have represented at cancer conferences in Canberra, and which was the prime mover in an effort to have the venue of the conference removed from Canberra to the various capital cities, has from its inception used every effort to make the campaign an Australian-wide one and to destroy any barrier which may have existed between the States. The Trust endeavoured to form an Australian cancer campaign and used every effort to bring something of this nature to fruition—it approached organizations, especially the Cancer Research Committee of the University of Sydney, and it approached prominent individuals in the hope that some organization or individual might take the initial steps to form a federal body. The result has been entirely negative. The Trust considered that the holding of an annual conference in the various capital cities in alternation would of itself be sufficient to draw attention to the need for a permanent and definite organization in each State, all of which could be coordinated to form an Australian cancer campaign. This not being acceptable, and the reasons which make Canberra a suitable and valuable place for meetings being fully realized, I, on behalf of the Trust, asked that arrangements be made for the holding of State conferences, supported by the Commonwealth Government.

The Trust does realize, and especially its representatives who have attended the conference at Canberra, its value in providing a meeting place free from social diversions for experts and others who are interested, and affording an opportunity for a review of the work which has been done during the year. It considers, however, that this deals only with one side of the line of attack upon the disease. The Trust was advised by Dr. Meyers, who as Secretary was collecting information for a Committee on Federal Organization, that, with the exception of work in Tasmania and Queensland, educational and publicity propaganda was not being carried out in any other State.

Surely this shows some vital weakness in the attitude of those who should be interested in the control of cancer. We are told that cancer may be cured by early treatment, yet I have heard it said by those interested in that treatment that if more publicity were given to the measures which were available for relief, it would not be possible to cope with the number of patients—an extraordinary argument to advance against the education of the public.

The Queensland Cancer Trust has realized that it is not sufficient to provide the means of treatment; people must be educated to take advantage of that treatment, and it must be made possible for them to reach it.

Publicity and educational work has been carried out throughout the State in all forms, including personal visits by representatives of the Trust to many centres annually. In addition, by an arrangement with the Government, which pays half the fare, free transportation to treatment centres is available for necessitous cases. Copies of all publicity matter issued by the Trust have been forwarded to the Commonwealth Department of Health and to cancer centres throughout the Commonwealth, with an offer to supply any number of copies which may be required with suitable headings at cost price. No advantage has been taken of this offer. We have before us the example of the British Empire Cancer Campaign and the American Society for the Control of Cancer, together with work undertaken by other organizations. Surely it is possible to arouse sufficient public interest to form an Australian organization on some such lines.

I trust I have said enough to convince you that the idea of holding State conferences was the opposite of "parochial".

Yours, etc.,
E. R. B. PIKE,
Secretary, Queensland Cancer Trust.

Ann Street,
Brisbane,
August 9, 1934.

LIFE.

SIR: The mental, athletic and emotional conditions in each human unit are governed in their expression to life by the three factors: health, training and ancestry.

A percentage of human minds, with a normal inborn inheritance and a favourable environment, may avoid degenerate habit. This may be attained through striving for a perfect expression of that type of effort which gives them the most intense and continuous interest. By so doing they guide and safeguard their development until maturity is reached. They can then increasingly endeavour to solve the problem of life.

An earnest effort day by day is necessary to the task until the early twilight of life: a twilight or dream

condition which has obscured their clarity of thought. They can then with confidence leave the bondsman's task to the active and receptive mind of youth.

Life should not linger when its task is done,
When faith is dim, when mind is on the wane,
Around it only fading hopes in view,
What profit then to look, and turn and strain
For something which the dark but still conceals.

It may be that a saving truth will come
To those whom in its reckless youth it drew
Out from the fertile Earth from whence it came,
To shield them from the doom of tainted years
With freedom from the stain of hopeless days.

A senile shade that creeps into the mind
Nor joy nor grief to break its tragic gloom,
The picture of a final phase in life,
With meaning only to an ageing mind,
Its dying colour drifting into night.

Yours, etc.,

G. H. TAYLOR.

1, Kirkoswald Avenue,
Mosman,
New South Wales,
August 22, 1934.

THE MORTALITY IN APPENDICITIS.

SIR: Mr. A. J. Trinca, in his criticism of my review on the mortality of appendicitis, gives his opinion, backed up, he says, by that of Mr. Sampson Handley, that the mortality in these cases is due to paralytic ileus. I would like to know what is the cause of the paralysis of the bowel, if not toxic action; also in what way paralytic ileus causes death, except through further toxæmia, more especially as he says that death in this disease is generally the result of delayed diagnosis and treatment. This delay is, after all, only fatal in that it allows the infection to spread to the general peritoneum or become more diffused locally, and the absorption of toxins from this greater area of infection increases the general and local toxic action.

I cannot understand Mr. Trinca's assertion that the drainage tubes of the surgeon do not drain away the fluids from the infected area. If this is the case, why are drain tubes universally used and what are they designed to drain from the peritoneal cavity? Moreover, the evidence of these cases proves that there is considerable drainage from the site of infection. This drainage is to remove the infection which has spread from the original site of infection, namely, the appendix, to the surrounding peritoneum.

It is realized, of course, that one of the dangers of any peritoneal infection is that due to the application of coils of intestine and omentum to the openings of the drainage tubes; they become blocked and pocketing of the infecting material occurs. Why are further surgical measures taken for drainage in these cases, if not to prevent further absorption of toxins? The drainage by the surgeon's tubes is to remove toxic material from the peritoneal cavity and prevent its local action and absorption into the general circulation, and this seems to be the only reason for drainage, and its efficiency is not a matter of opinion, but of fact, proved by universal usage, and many have been the calamities which have resulted from failing to use this generally accepted technique: as the area of toxin formation is not localized to the immediate openings of the drainage tube, if the toxic material can make its way to the drainage openings, antitoxic serum poured down the drainage tube can make its way to the areas of infection. Actual experience has proved that this is the case.

If we accept Mr. Trinca's views, the universally accepted methods of dealing with peritoneal infection, by drainage tubes, is wrong. Moreover, if drainage is not instituted, the infection which cannot be removed at the time of operation is left to be dealt with by the bacteriolytic and antitoxic forces of the body. As I have said in my previous review, no surgeon can gauge these defences, and no one, however hopeful, would rely on these alone in the face of severe local or general peritoneal infection.

I do not think, although I have due respect for Mr. Trinca's views, that I will neglect adequate drainage in the future.

As regards the antitoxic serum, which I introduce into the peritoneal cavity, this is a specific antitoxic serum.

The final paragraph in Mr. Trinca's article has, I consider, lost its point in the face of what I have already said.

Yours, etc.,

EDWARD HENTY SMALPAGE,
M.B., Ch.M. (Syd.), F.R.C.S. (Eng.).

"Hengrove Hall",
193, Macquarie Street,
Sydney,
August 19, 1934.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on August 13, 1934.

The degree of Bachelor of Surgery (B.S.) was conferred *in absentia* on Robert Samuel Irwin (Maclean, New South Wales).

The Diploma in Tropical Medicine was awarded to Robert William Cooper, M.D., B.S. (Melbourne), Carl Ernest Mitchelmore Gunther, M.B., B.S. (Sydney), George Hugh McQueen, M.B., B.S. (Melbourne), Bruce Robson Overend, M.B., Ch.M. (Sydney), and Frederick Jones Williams, M.B., B.S. (Melbourne).

A gift of £52 was accepted with grateful thanks from Dr. and Mrs. A. A. Pain for the establishment of an annual prize in geology to commemorate the work of Emeritus Professor Sir Edgeworth David.

The appointment of Major E. H. Booth, M.C., B.Sc., as Secretary of the University Extension Board was approved.

Professor J. C. Earl, Professor L. A. Cotton, and Professor T. G. B. Osborn were appointed as delegates to the meeting of the Council of the Australian and New Zealand Association for the Advancement of Science, to be held in Melbourne in January next.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Russell, Walter McRae, M.B., B.S., 1918 (Univ. Melbourne), Bellerive, Tasmania.

Obituary.

HARWOOD HENRY OSBORN.

WE regret to announce the death of Dr. Harwood Henry Osborn, which occurred on August 19, 1934, at Toorak, Victoria.

FRANCIS WILLIAM KANE.

WE regret to announce the death of Dr. Francis William Kane, which occurred on August 24, 1934, at Dulwich Hill, New South Wales.

ANDREW NORRIE.

WE regret to announce the death of Dr. Andrew Norrie, which occurred on August 25, 1934, at Chatswood, New South Wales.

Books Received.

THE SCIENCE OF SIGNS AND SYMPTOMS IN RELATION TO MODERN DIAGNOSIS AND TREATMENT: A TEXT-BOOK FOR GENERAL PRACTITIONERS OF MEDICINE, by R. J. S. McDowall, D.Sc., M.B., F.R.C.P., with the assistance of H. A. Dunlop, M.D., M.R.C.P.; Third Edition; 1934. London: William Heinemann (Medical Books) Limited. Royal 8vo., pp. 557, with illustrations. Price: 21s. net.

Diary for the Month.

SEPT. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 4.—Tasmanian Branch, B.M.A.: Council.
SEPT. 5.—Western Australian Branch, B.M.A.: Council.
SEPT. 5.—Victorian Branch, B.M.A.: Branch.
SEPT. 6.—South Australian Branch, B.M.A.: Council.
SEPT. 7.—Queensland Branch, B.M.A.: Branch.
SEPT. 11.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 14.—Queensland Branch, B.M.A.: Council.
SEPT. 18.—Tasmanian Branch, B.M.A.: Council.
SEPT. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 19.—Western Australian Branch, B.M.A.: Branch.
SEPT. 19.—Victorian Branch, B.M.A.: Clinical Meeting.
SEPT. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 26.—Victorian Branch, B.M.A.: Council.
SEPT. 27.—South Australian Branch, B.M.A.: Branch.
SEPT. 27.—New South Wales Branch, B.M.A.: Branch.
SEPT. 28.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of the Affiliated Local Associations of Members with the Council.

Medical Appointments.

Dr. L. M. T. Hungerford has been appointed Quarantine Officer at Esperance, Western Australia, under the *Quarantine Act*, 1908-1924.

* * *

Dr. N. McColl (B.M.A.) has been appointed a Certifying Medical Practitioner at Maffra, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. D. Lewis has been appointed Medical Officer of Health by the Westonia Road Board, Western Australia.

* * *

Dr. E. K. Wallace has been appointed a Resident Medical Officer at the Adelaide Hospital, South Australia.

* * *

Dr. J. J. Searby (B.M.A.) has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be a Certifying Medical Practitioner at Ringwood, Victoria.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi, xvii and xix.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, New South Wales: Resident Medical Officers.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr. Innisfail Hospitals Board.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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